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Med.

HEART.

I

A JOURNAL FOR THE STUDY OF THE CIRCULATION.

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Page 358. Line 6 from the bottom, for “—” read “+”.

Page 361. The columns in the table are “Cycle,” “Systole,” and
“K” from left to right.

Page 368. Case No. 36 under K, for “0.478” read “0.378.”

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ORTHODIAGRAPHIC OBSERVATIONS ON THE SIZE OF THE
HEART IN CASES OF SO-CALLED "IRRITABLE
HEART."*

BY J. C. MEAKINS AND E. B. GUNSON.

(From the Military Heart Hospital, Hampstead.)

IN such a condition as that of so-called "irritable heart" (the symptom-complex of dyspnoea, palpitation, precordial pain, dizziness and fatigue), where certain of the cardinal symptoms are usually referred to the heart, it is of importance to study the size of this organ under conditions of rest and strain. The usual clinical methods of estimating this are not sufficiently accurate to detect small but important changes from the normality. So in order to eliminate these errors the following observations were carried out with an orthodiagraph.

Methods.

The Middlesex orthodiagraph was employed. With a little practice it was used with rapidity and accuracy. The possible sources of error of position lay in the three objects concerned, namely, the patient, the X-Ray table and the paper upon which the diagrams were made. It was found that these three should always be in exactly the same relationship to each other. Therefore the following precautions were necessary.

(a.) *The patient.*—Certain definite landmarks were outlined with material which would be plainly recognizable with the X-Ray. 1. A narrow strip of lead was fixed with adhesive plaster so as to extend from the sixth cervical spinous process to that of the eighth dorsal (Fig. 1). 2. A thick piece of copper wire was fixed down the middle of the sternum from the

* Observations carried out on behalf of the Royal Medical Research Committee.

supra-sternal notch to near the umbilicus passing over the centre of the ensiform cartilage. 3. A cross-wire was placed over the junction of the manubrium and the sternum. 4. Another cross-wire was fixed across the sternum at the level of the fourth intercostal space (Fig. 2). A small circle of wire was placed over each nipple and a cross over the maximal apex beat.

(b.) *The X-Ray table.*—1. A piece of flattened wire was firmly fixed down the centre of the table (Fig. 3 A). 2. A similar piece of wire was placed at right angles to this twenty-four inches from the head of the table (Fig. 3 B). 3. A longer cross-wire was placed four inches below this (Fig. 3 C).

(c.) *The recording paper.*—The orthodiagraph was moved upon a large piece of thick plate glass. In order that the wheels should not run over the recording paper, this was raised from the glass by a piece of cardboard exactly the same size as the paper, *i.e.*, 14 by 8½ inches. This cardboard was firmly glued to the glass so that its long diameter was at right angles to the long diameter of the table and the centre of the card was directly under the middle line of the table and centred relative to the cross-wires (Fig. 3 B and C).

Following these adjustments the patient was placed in the recumbent position so that the strip of lead over the spine was as nearly as possible superimposed on the longitudinal wire on the table and the upper cross-wire over the sternum was in the same plane as and vertically above the upper cross-wire on the table. He was then examined by the X-Ray and these two relations were confirmed and corrected if necessary. Finally he was ordered to rotate the chest either to the right or left until the longitudinal wire on the sternum was accurately over the lead strip on the spinous processes and the longitudinal wire on the table. It was taken that the patient was centred if these three lines were superimposed so as to show but one shadow in their entire length, if the upper cross-wires on the sternum and the table also showed but one shadow, and lastly, if the lower cross-wires were parallel.

If all these precautions are taken orthodiagrams can be obtained from the same patient on the same day or on successive days with practically identical results (see Fig. 4, which is an example of tracings reduced to half natural size).* A maximum error of no more than 0.5 cm. is allowed in the total transverse measurement of the heart. In order that errors from respiration should be eliminated, the points marked A, C, D, E and F in Fig. 3 should always coincide in different tracings from the same patient,

* In order to tabulate the findings as simply as possible only two measurements are used. R.M., or right margin, was from the mid-sternal line to the outer limit of the right border of the heart. L.M., or left margin, from the mid-sternal line or extension thereof to the outer limit of the left border of the heart.

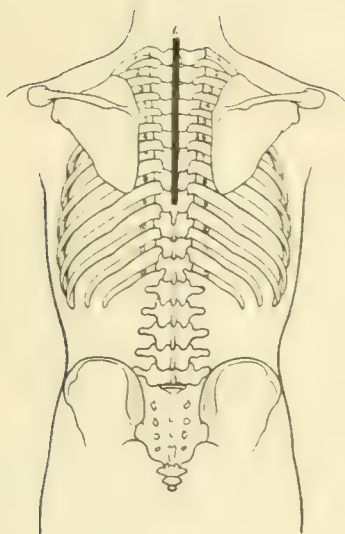


Fig. 1.

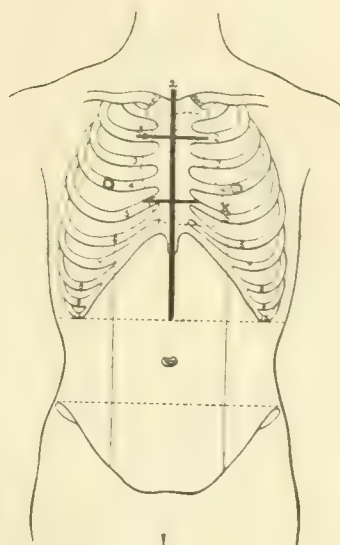


Fig. 2.

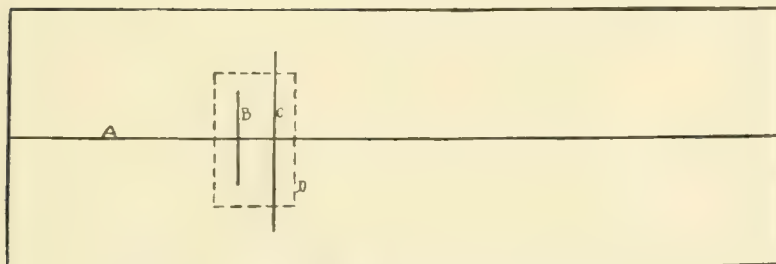


Fig. 3.

and the lines A-B should always superimpose one on the other. Inconstancy in the position of B, relative to A, is unavoidable in some cases (as in Fig. 3), but such inconstancy, if not great, is immaterial. Furthermore the point c (left chest wall) should always be the same distance from the line A-B in the same patient, and similarly the point d (right chest wall). In many patients the two sides of the chest are not equal, but the diagram should show constant asymmetry for the same person from time to time.

The following table will demonstrate the accuracy with which measurements may be obtained from time to time in one and the same case* (Table I).

TABLE I.

Name.	Date.	R.M. (cm.)	L.M. (cm.)	Total. (cm.)
McG.	August 10	3.1	7.7	10.8
	„ 14	3.2	7.9	11.1
P.	August 18	4.3	9.0	13.3
	„ 21	4.4	9.0	13.4
G.	July 17	3.9	9.4	13.3
	„ 21	3.7	9.4	13.1
S.	July 19	4.4	7.2	11.6
	„ 28	4.1	7.5	11.6
F.	July 20	4.2	7.5	11.7
	„ 26	3.8	7.9	11.7
C.	August 7	4.3	7.9	12.2
	„ 10	4.5	8.0	12.5
M.	August 9	4.2	7.7	11.9
	„ 10	4.0	8.0	12.0
T.	June 28	4.2	8.5	12.7
	„ 29	4.3	8.2	12.5
	July 15	4.2	8.5	12.7
O'R.	June 29	4.7	9.2	13.9
	July 15	4.7	9.0	13.7
	August 8	4.6	9.4	14.0
H.	June 28	4.2	10.7	14.9
	„ 29	4.2	10.7	14.9
	July 16	4.7	10.2	14.9

(d.) *Effect of quiet respiration on measurements of the heart.*—It was deemed necessary to determine what effect the position of the diaphragm would have on the measurements of the heart. In order to do this six cases were examined and measurements were recorded at the end of quiet inspiration and expiration. The results are found in Table II.

* All the measurements recorded in the remaining tables are based upon at least duplicate observations and the averages taken.

TABLE II.
EFFECT OF RESPIRATION ON MEASUREMENTS OF THE HEART.

Name.	Respiratory period.	R.M. (cm.)	L. M. (cm.)	TOTAL. (cm.)
Sf.	Expiration	3.8	7.9	11.7
	Inspiration	4.0	7.6	11.6
M.	Expiration	2.9	8.9	11.8
	Inspiration	2.8	8.9	11.7
F.	Expiration	3.7	7.9	11.6
	Inspiration	3.8	7.5	11.3
C.	Expiration	4.5	7.9	12.4
	Inspiration	4.4	8.1	12.5
N.	Expiration	4.1	7.9	12.0
	Inspiration	4.1	7.9	12.0
R.	Expiration	3.5	8.7	12.2
	Inspiration	3.3	8.8	12.1

It is apparent that little or no change in the measurement occurs during quiet breathing. In forced respiration a greater difference would be looked for. This in fact does occur at times, but it is not at all constant and averages less than 0.5 cm. in the total transverse measurement of the heart. As a routine points were marked during the expiratory pause of quiet respiration.

The size of the heart in cases of so-called "irritable heart."

Fifty patients suffering from typical symptoms of this condition were examined by the orthodiagraph to determine the size of the heart relative to that of healthy men. There was selection inasmuch as cases of mitral stenosis, aortic regurgitation, aneurism, auricular flutter and auricular fibrillation were excluded. The selection did not exclude cases in which enlargement was thought to exist before examination. All the cases were examined twice, the greatest divergence of the total width in any case being 0.3 cm.. The average values were adopted. It has been found in the normal individual that the size of the heart, *i.e.*, the sum of the greatest distance between the mid line and the right and left border, is in direct ratio to the body weight. T. Dietlen¹ has recorded the size of the heart in 184 normal adult males, including 59 young soldiers. These measurements are taken here as the standard of normal cases.

The measurements of 50 cases of so-called "irritable heart" are given in Table III.

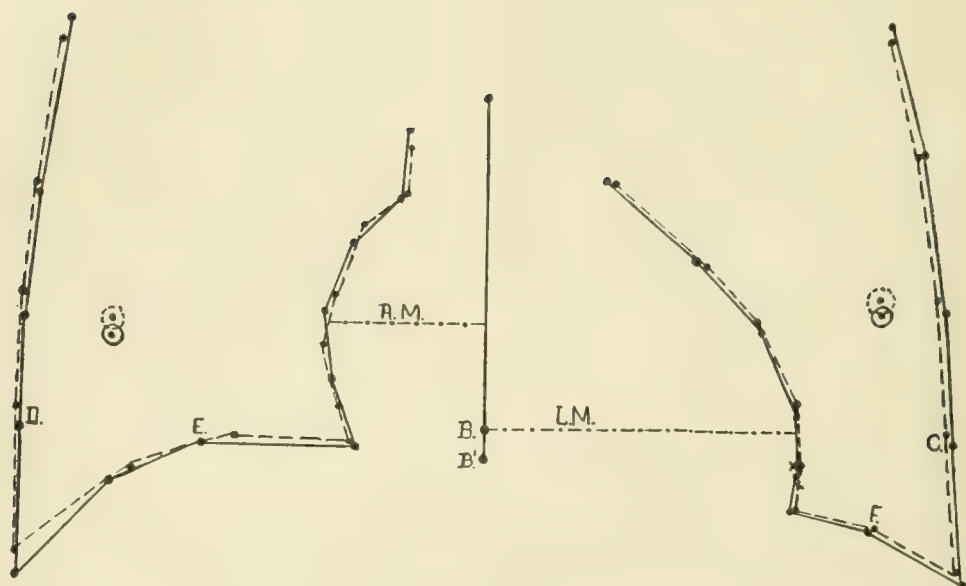


Fig. 4.

The average size of the heart in these cases departs very little from the normal. In this condition cardiac enlargement was rarely observed. In fact in only two of these cases (4 per cent.) was there an enlargement of more than 0.5 cm. over the normal average, as stated by Dietlen, and in both of these exceptions it amounted to 0.7 cm. (Table III). On the other hand in over 60 per cent. of these cases the heart was smaller than the average of controls by more than 0.5 cm.. In all cases the records were taken from patients who were up and about the wards and grounds of the hospital.

Such differences as may be found between the diameters (average) of this series and that of Dietlen are small; such as they are they are sufficiently accounted for by (a) slight increase of pulse rate in this series as compared to the normal (77.5); (b) the fact that a large percentage of these men had been in sedentary occupations for the greater part of their lives.

TABLE III.

Body weight.	Patient.	Pulse	R.M. (cm.)	L.M. (cm.)	Total. (cm.)	Patients' average. (cm.)	Dietlen's average.* (cm.)
118	C.	84	3.7	8.1	11.8	11.4	12.4
118	M.	92	3.4	9.2	12.6		
119	G.	77	3.6	7.3	10.9		
119	McG.	68	3.3	7.2	10.5		
126	A.	72	3.8	8.7	12.5	12.2	12.0
131	C.	80	3.1	9.2	12.3		
132	D.	60	5.7	7.6	13.3		
132	K.	100	4.0	7.2	11.2		
133	P.	72	4.4	7.7	12.1		
133	W.	80	3.9	7.9	11.8		
134	S.	104	4.5	7.6	12.1		
135	K.	64	4.0	8.5	12.5	12.1	13.1
135	N.	68	4.4	7.6	12.0		
135	W.	64	4.3	7.0	11.3		
136	C.	72	5.1	6.7	11.8		
137	E.	88	4.2	7.5	11.7		
137	T.	76	4.2	8.3	12.5		
138	H.	70	4.9	7.0	11.9		
139	S.	84	4.2	6.8	11.0		
139	W.	80	4.3	8.2	12.5		
140	B.	64	4.5	8.4	12.9		
140	P.	90	3.0	9.7	12.7		
140	R.	72	3.5	8.9	12.4		
141	B.	76	3.6	7.8	11.4		
141	C.	76	4.2	7.4	11.6		
142	O'C.	90	4.0	7.9	11.9		
144	L.	100	4.8	7.1	11.9		
144	M.	72	4.8	8.8	13.6		
145	W.	92	3.3	10.0	13.3		
147	B.	68	4.8	8.2	13.0	12.5	13.2
148	M.	80	3.8	9.0	12.8		
148	M.	104	3.6	9.1	12.7		
148	W.	84	4.6	6.8	11.4		
150	B.	80	5.7	6.1	11.8		
150	R.	72	3.7	8.7	12.4		
151	B.	72	3.4	9.7	13.1		
154	D.	70	3.7	9.4	13.1		
155	G.	66	3.4	9.0	12.4		
157	G.	90	4.5	8.2	12.7		
159	T.	57	4.5	9.3	13.8	13.9	13.4
160	O'R.	72	4.8	9.1	13.9		
162	G.	68	4.4	9.3	13.7		
*163	H.	64	4.2	9.9	14.1		
169	C.	96	4.4	8.6	13.0	13.7	14.3
169	W.	80	5.1	8.3	13.4		
170	B.	64	3.0	10.2	13.2		
*172	H.	62	4.0	11.0	15.0		
174	J.	92	3.7	9.6	13.3		
181	R.	76	5.0	7.8	12.8	13.3	14.4
186	S.	71	4.0	9.7	13.7		
						12.7	13.4

* These averages are taken from Dietlen's tables, and represent the average measurements of his cases grouped according to body weight in kilograms.

Four cases of so-called "irritable heart" which appeared to have some enlargement of the heart to percussion were examined repeatedly (three cases five times, and one case four times) in order to be absolutely certain as to the size of the heart. The average results are shown in Table IV.

TABLE IV.

Name.	Pulse.	R.M. (cm.)	L.M. (cm.)	Patients' total. (cm.)	Normal for same body weight. (cm.)
T.	76	4.3 (3.0)	8.5 (14.0)	12.8 (17.0)	13.1
O'R.	70	4.6 (4.0)	9.5 (13.0)	14.1 (17.0)	13.8
H.	56	4.25 (3.5)	10.75 (13.0)	15.0 (16.5)	14.3
D.	69	3.8 (4.0)	9.6 (13.0)	13.4 (17.0)	13.4
Average				13.8	13.7

In three of these cases (T., O'R. and D.) the average variation from the normal orthodiagraph measurements for their body weight is negligible. In the case of H. there is a slight increase (0.7 cm.) over the normal.

On comparing the percussion measurements (in parentheses in Table IV) with the orthodiagraph measurements, two points of importance are to be noted. The right border (R.M.) on percussion is, as a rule, farther in than that found with the orthodiagraph, while the left border (L.M.) on percussion is from 2.25 cm. to 5.5 cm. farther out than the orthodiagraph measurements. The total percussion measurement of the heart in all four cases is greater than that found with the orthodiagraph. This discrepancy between the percussion and orthodiagraph measurements is found in 70 per cent. of the cases examined. It is of importance in view of the fact that there is commonly supposed to be an increase of the cardiac dullness in this condition, and therefore an enlargement of the heart has been inferred. This, in our experience, is contrary to fact, as we have found no evidence with the orthodiagraph of any appreciable enlargement even in many of those cases where percussion would lead one to suppose such an enlargement to be present.

In about 20 per cent. of cases of so-called "irritable heart" there is a diffuse apical impulse which may extend over two, three, or more, rib spaces. It was considered of importance to determine if there were any evidence

of enlargement of the heart in such cases. Out of 50 cases examined with the orthodiagraph there were nine with a diffuse apical impulse. These cases are shown in Table V.

TABLE V.

Name.	Pulse.	Impulse.	R.M. (cm.)	L.M. (cm.)	Patients' total. (cm.)	Normal average. (cm.)
O'R.	72	Diffuse 3 spaces	4.6	9.5	14.1	13.8
D.	72	.. 2 ..	3.8	9.6	13.4	13.4
W.	64	.. 3 ..	4.3	7.0	11.3	13.1
B.	64	.. 3 ..	3.0	10.2	13.2	14.3
O'C.	80	.. 3 ..	4.0	7.9	11.9	13.1
B.	72	.. general	3.6	7.8	11.4	13.1
B.	68	.. 2 spaces	4.8	8.2	13.0	13.2
G.	68	.. 3 ..	4.4	9.3	13.7	14.3
R.	72	.. 4 ..	3.7	8.7	12.4	13.2
Average					12.7	13.5

In no case is the size of the heart appreciably greater than normal. In fact the average of these nine cases shows a proportionate decrease (0.8 cm.) which is identical to the decrease found in the 41 cases (0.7 cm.) without a diffuse apical impulse.

Effect of rest on the size of the heart in the same cases.

It has been noted in regard to the pulse rate and symptomatology that rest in bed has a distinctly deleterious effect upon patients suffering from the condition considered. Therefore it was of importance to determine what influence rest in bed might have on the size of the heart in these cases.

The effect of twelve hours rest was first investigated, that is to say, a comparison was instituted between the size of the heart at 9 a.m. and before the patient had been out of bed (having gone to bed at 9 p.m.) and at 3 p.m., after he had been up during the day and had done in the majority of cases (five out of six) one half-hour's Swedish drill during the morning. The results are found in Table VI.

The greatest difference between the morning and afternoon transverse measurements of the heart is 0.3 cm., which is well within the normal margin of error.

TABLE VI.

Name.	Time.	Pulse.	Transverse measurement. (cm.)
McG.	9 a.m.	70	10.9
	3 p.m.	60	10.9
S.	9 a.m.	77	11.8
	3 p.m.	70	11.5
W.	9 a.m.	78	11.6
	3 p.m.	80	11.7
McB.	9 a.m.	60	11.6
	3 p.m.	65	11.5
Nr.	9 a.m.	72	12.7
	3 p.m.	74	12.7
N.	9 a.m.	76	13.7
	3 p.m.	78	13.5

In order to further test the effect of rest, seven patients were put to bed for periods varying from seven to fourteen days. The size of the heart was determined before the patient was put to bed and afterwards. The results are shown in Table VII.

TABLE VII.

Name.	Date.	Pulse rate.	Transverse measurement. (cm.)	Conditions as to rest.	Change in size of heart. (cm.)
S.	June 27	80	11.2	Out of bed	
	July 4	80	12.8	In bed 7 days	1.2 +
T.	July 7	57	13.9	Out of bed	
	„ 15	68	14.0	In bed 8 days	0.1 +
C.	July 7	125	11.8	Out of bed	
	„ 16	82	12.8	In bed 9 days	1.0 +
O.	July 15	69	12.4	Out of bed	
	„ 24	74	13.0	In bed 9 days	0.6 +
Ta.	Feb. 17.17	70	12.7	Out of bed	
	Mar. 3.17	68	13.3	In bed 14 days	0.6 +
B.	Feb. 17.17	82	11.0	Out of bed	
	Mar. 3.17	80	11.9	In bed 14 days	0.9 +
D.	Feb. 17.17	70	11.0	Out of bed	
	Mar. 3.17	72	11.6	In bed 14 days	0.6 +

In all seven cases there is an increase in the transverse measurement of the heart. The minimum increase is 0.1 cm., and the maximum is 1.2 cm., the average being 0.7 cm.. In six of the seven cases the change in size is greater than 0.5 cm..

The effect of exercise on the size of the heart in the same patients.

In healthy men the heart is smaller after exercise than before it, according to De la Camp, Moritz, Nicolai and Zuntz², and others. As to change during exercise few observations have been made, but Nicolai and Zuntz found a quite negligible increase in size in four cases. A paper by C. S. Williams³ has recently come to our notice. This paper reviews very fully previous observations upon the size of the heart in exercise, and adds new observations of the heart's diameter in health and disease by the telercöntgen method. This observer finds a decrease in the size of the normal heart immediately after strenuous exercise in twenty-seven out of thirty-three subjects. In the remaining six instances the change was negligible, being no more than a millimetre one way or the other. In certain conditions, *i.e.*, structural heart disease, pulmonary tuberculosis, he has found dilatation after exercise on occasion; but with equal frequency he has found contraction in the same conditions.

In order to determine what change, if any, occurs in the size of the heart during exercise in cases of so-called "irritable heart," several series of orthodiagraphic observations were made under varying conditions. The first series was as follows:—The patient was placed in the horizontal position on the examining table and the pelvis steadied by means of a circular strap attached to the table; precautions for accurately centring the patient were observed. Orthodiagrams were then taken, and the pulse rate noted. The patient then raised each leg, alternately, sixteen times per minute, to a height of about three feet from the table, keeping the body as steady as possible. The effect of the exercise was, after three or four minutes, to raise the pulse rate to above 100 per minute, as a rule to about 120, and to produce conspicuous increase in respiratory rate, followed towards the end of the observations by considerable fatigue in most cases.

When the pulse rate had risen approximately to 120 per minute an orthodiagram was taken, the patient continuing to exercise and counting aloud to obviate fixation of the diaphragm. Four to seven minutes after the exercise had ceased and the pulse rate returned to normal another record was made. As very slight decrease in the transverse diameter of the heart occurs in quiet breathing during inspiration (see Table II), attributable directly to the lowered position of the diaphragm, records were made both in the inspiratory and expiratory positions, and comparative measurements made with the diaphragm approximately in the same position.

The simple movement of raising the leg was found to produce a lateral movement of the whole chest, including the heart, of from 0.5 to 1 cm.; especially was the movement marked when fatigue was induced. The possible error arising from this cause is such as largely to invalidate any orthodiagraph findings in which the changes are of the small order of magnitude obtained during the present investigations, and any observations during exercise other than those obtained by instantaneous photographs must be open to the same criticism.

The results obtained in eight cases are set out in Table VIII. After an allowance of 0.5 cm. is made for errors of technique, in no case is there a recorded change in the size of the heart during this exercise.

TABLE VIII.

Name.	Relation to exercise.	Pulse.	R.M. (cm.)	L.M. (cm.)	Total. (cm.)
C.	Before	69	4.5	8.0	12.5
	During	106	4.1	8.4	12.5
	After	66	5.0	8.0	13.0
N.	Before	63	4.1	7.9	12.0
	During	102	4.1	7.7	11.8
	After	62	4.1	8.4	12.5
R.	Before	76	3.5	8.7	12.2
	During	108	3.8	8.3	12.1
	After	69	3.5	8.8	12.3
Sp.	Before	71	4.1	7.7	11.8
	During	119	4.1	7.7	11.6
	After	70	4.2	7.6	11.8
M.	Before	90	3.1	9.1	12.2
	During	119	3.3	8.8	12.1
	After	77	3.3	8.9	12.2
F.	Before	80	4.0	7.7	11.7
	During	114	4.2	7.8	12.0
	After	78	3.8	7.8	11.6
B.	Before	68	4.1	8.9	13.0
	During	116	4.2	8.5	12.7
	After	64	4.0	9.0	13.0
G.	Before	60	3.8	9.4	13.2
	During	119	3.8	8.9	12.7
	After	60	3.8	9.1	12.9

As the movement of the trunk during the exercise of raising the legs from the table may interfere with the accuracy of the results, it was decided to repeat the experiments, using a different form of exercise. It seemed impossible to obtain an exercise sufficiently severe to produce symptoms and at the same time an exercise free from the defects alluded to, so records during the actual period of work were no longer attempted. In order that accurate observations may be made immediately exertion ceases, it is essential that a symmetrical exercise be used which will allow of the trunk being in exactly the same position during all the observations. This was accomplished by accurately centring the patient and then fixing the thorax and hips as far as possible by the use of sand-bags on either side. The exercise adopted was the raising of 10-pound dumbbells. One was placed in each hand and the patient was instructed to swing the arms outwards to their full extent and then back to the chest at the rate of twenty to the

minute. He was instructed to persist in this until fatigued or symptoms become pronounced. It may be stated that in this exercise local fatigue of the arms almost always occurred before the breathlessness became at all distressing. The first observations were made within as short a time as possible after the exercise had ceased. This was accomplished within ten seconds of the cessation of effort, and the first points recorded were the outside limits of the heart to the right and to the left. It may be stated that there was a definite acceleration of the heart action immediately after the exercise, as this could be readily observed under the fluoroscope.

TABLE IX.

No.	Name	When examined.	R.M. (cm.)	L.M. (cm.)	Total. (cm.)	Change (cm.)
1.	C.	Before ex. After ex.	3.9 3.6	8.8 9.0	12.7 12.6	0.1—
2.	W.	Before ex. After ex.	4.3 3.8	7.4 8.0	11.7 11.8	0.1+
3.	M.	Before ex. After ex.	4.2 3.8	6.7 7.3	10.9 11.1	0.2 +
4.	N.	Before ex. After ex.	3.3 3.3	9.8 10.0	13.1 13.3	0.2+
5.	E.	Before ex. After ex.	3.9 3.9	7.0 7.1	10.9 11.0	0.1+
6.	Wr.	Before ex. After ex.	4.1 4.1	8.8 8.9	12.9 13.0	0.1+
7.	McB.	Before ex. After ex.	3.3 4.0	8.0 7.4	11.3 11.4	0.1 .

Seven cases were examined in this way and the results are recorded in Table IX. The figures show practically no change in the size of the heart immediately after this exercise, the greatest variation being only 0.2 cm..

Therefore it may be concluded that with such exercises as the above there is no change in the size of the heart in these cases.

Effects of Swedish drill upon the size of the heart in the same cases.

The exercises so far described were of a comparatively mild type and of short duration; those now to be considered were more prolonged and strenuous. The cases were examined from two standpoints—first for differences in the size of the heart after a period of comparative rest and after some weeks of daily Swedish drill; secondly, for change in the size of the heart immediately after one half-hour's strenuous physical exertion.

Results of daily exercise. Nine patients were allowed up and about in the wards, but were not allowed outside the building, and were given no physical exercises. At the end of two weeks orthodiagrams were taken, and then the patients were placed on daily physical exercises, which were gradually increased in severity, until half-an-hour of Swedish drill was done each day, when another outline of the heart was recorded. The results are shown in Table X. It will be observed that any change in the size of the hearts over this period is well within the usual limits of error. Thus it may be said that there is no appreciable change in the size of the heart in these cases as a result of the continued and daily physical exercises.

TABLE X.

No.	Time.	Pulse.	R.M. (cm.)	L.M. (cm.)	Total. (cm.)	Change. (cm.)
1.	After 2 weeks rest	74	3.6	8.4	12.0	0.1 +
	„ 6 „ exercise	78	3.6	8.5	12.1	
2.	After 2 weeks rest	86	4.5	7.2	11.7	0.4 —
	„ 6 „ exercise	104	4.5	6.8	11.3	
3.	After 2 weeks rest	65	4.2	7.8	12.0	0.1 +
	„ 6 „ exercise	62	4.0	8.1	12.1	
4.	After 2 weeks rest	82	3.6	8.9	12.5	0.1 —
	„ 6 „ exercise	76	3.9	8.5	12.4	
5.	After 2 weeks rest	64	3.6	7.8	11.4	0.4 +
	„ 6 „ exercise	66	3.8	8.0	11.8	
6.	After 2 weeks rest	90	4.0	7.9	11.9	0.3 +
	„ 6 „ exercise	80	4.4	7.8	12.2	
7.	After 2 weeks rest	60	4.6	8.6	13.2	0.1 +
	„ 6 „ exercise	72	5.1	8.2	13.3	
8.	After 2 weeks rest	78	4.0	8.7	12.7	0.1 +
	„ 6 „ exercise	86	4.1	8.7	12.8	
9.	After 2 weeks rest	72	2.4	10.8	13.2	0.5 +
	„ 6 „ exercise	80	3.0	10.7	13.7	

Size of the heart immediately after Swedish exercises. Eleven cases of varying degrees of severity were examined with the orthodiagraph to determine if there is any change in the size of the heart immediately after Swedish exercises. Five cases were on D.30, three on C.30, one on B.C.30, and two on C.15.* A careful note was taken at the time as to the symptoms produced by the exercise. An orthodiagram was taken before the patient did the drill and another immediately afterwards. The latter was taken as soon as possible after the cessation of exertion, and the right and left borders of the heart

* The letters refer to the grade of Swedish exercise and the numerals to the duration in minutes. They are given in the order of their severity, D.30 being most severe, C.15 least severe.

were always ascertained within the first half minute. The pulse and respiratory rates were not taken, as this would have necessitated a certain delay in taking the orthodiagram after the exercise. The heart's action and respiratory rate could be very easily determined on the screen, and it was constantly observed that the more severe the symptoms the more rapid were they. The results of these observations are shown in Table XI.

TABLE XI.

No.	Name.	When examined.	R.M. (cm.)	L.M. (cm.)	Total. (cm.)	Change. (cm.)	Symptoms during exercise.			
							Palp.	Dys.	Vert.	Pain.
1.	McG.	Before	3.0	7.9	10.9	1.1—	—	—	—	—
		After D.30	2.6	7.2	9.8					
2.	S.	Before	4.3	7.5	11.8	1.1—	—	—	—	—
		After D.30	3.5	7.2	10.7					
3.	N.	Before	4.0	9.7	13.7	1.0—	—	—	—	—
		After C.30	3.6	9.1	12.7					
4.	McB.	Before	3.6	8.0	11.6	1.2—	—	—	—	—
		After C.30	3.5	6.9	10.4					
5.	P.	Before	4.4	9.0	13.4	0.8—	sl.	sl.	—	—
		After D.30	4.1	8.5	12.6					
6.	C.	Before	2.6	9.6	12.2	0.6—	sl.	sl.	sl.	—
		After C.30	2.6	9.0	11.6					
7.	F.	Before	3.9	8.7	12.6	0.2+	+	+	+	+
		After D.30	3.8	9.0	12.8					
8.	E.	Before	4.0	8.8	12.8	0.2—	+	+	+	+
		After D.30	3.8	8.8	12.6					
9.	Wi.	Before	4.0	7.6	11.6	0.2—	+	+	—	+
		After C.15	3.0	8.4	11.4					
10.	Wr.	Before	4.0	8.6	12.6	0.2+	+	+	+	+
		After C.15	4.3	8.5	12.8					
11.	D.	Before	3.5	9.7	13.2	0.4—	+	+	—	—
		After B.C.	3.9	9.7	13.6					

These cases apparently fall into two distinct groups in regard to the size of the heart after exercise. In one group there is a distinct diminution in the cardiac diameter amounting to 1 cm. or more. There are four such cases and two less conspicuous cases. The other six cases show negligible changes. A further difference between these two groups is established by the presence or absence of symptoms.

In the first four cases in which there was a distinct reduction in the size of the heart there were no appreciable symptoms despite the severity of the exercises. In *CASES* 5 and 6 there were slight symptoms, and at the same time the reductions in size were not so conspicuous as in the first four cases. *CASES* 7 to 11 experienced considerable symptoms on the

exercises. In two cases there was an insignificant diminution of the measurement (0.2 cm.), and in two cases a similar increase of the measurement. In *CASE 11* there was a somewhat greater increase in the measurement, and this patient was compelled to fall out at the exercise on account of the severity of the symptoms.

Such variation as was found in the last four cases of this table fall within the error of measurement. In no case can it be said that there was an increase in the size of the heart, though such may be suspected in *CASE 11*. The contrast is between the first group in which there was a definite decrease in size, and the second group in which the size remained more or less constant.

The differences in the reaction of the two groups is not to be explained by variation in the reaction of the heart rate, for increased heart rate diminishes the size of the organ, and acceleration was more to be noted in *CASES 6 to 11*.

CONCLUSIONS.

1. The heart in cases of so-called "irritable heart" is, on the average, somewhat smaller (0.7 cm.) than normal.

2. In cases with a diffuse apical impulse no enlargement is shown by the orthodiagraph. On the contrary, the average measurement is smaller than the normal, in the same proportion as in those who do not exhibit this sign.

3. When cases of so-called "irritable heart" rest in bed there is an average increase in the transverse diameter of the heart of 0.7 cm..

4. After strenuous Swedish exercise in cases having no material symptoms there is a decrease (1 cm.) in the size of the heart, while in cases showing conspicuous symptoms there is, on the average, no appreciable change in the size of the heart.

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THE EFFECT OF CERTAIN SENSORY STIMULATIONS ON
RESPIRATORY AND HEART RATE IN CASES OF SO-CALLED
“IRRITABLE HEART.”*

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IN patients suffering from so-called “irritable heart” (the chief complaints being shortness of breath, palpitation, precordial pain, dizziness and fatigue), the increase of the pulse and respiratory rate on exertion is pronounced and has been dealt with in other communications from this hospital. The similar effect of stimuli calculated to give rise to emotion is frequently complained of by the patients. In fact, palpitation while under bombardment may be so distressing as to incapacitate a soldier. Associated with prolonged palpitation produced in this manner precordial pain may become very severe and be bitterly complained of. In order to ascertain the response of the pulse and respiratory rate in these patients to stimulation producing emotional changes the following observations were undertaken.

Method. As excitement can easily be produced by visual or auditory stimuli, these were employed. As a visual stimulus an unexpected flame in a darkened room is used. A heaped teaspoonful of a mixture of equal parts of potassium chlorate and sugar is placed in a pan within the range of vision. To this 1 c.c. of sulphuric acid is added at the right moment. A bright flame shoots up suddenly some two feet in the air and lasts about three seconds. As an auditory stimulus the unexpected discharge of a blank cartridge under the examining couch is used. Both patients and controls are in the recumbent position for some minutes before stimulation. A continuous graphic record of the respiration and pulse are taken during this period and continued throughout the test and for some while afterwards.

Response of controls. Six healthy subjects were used as controls. They were ignorant of the intended stimulation, although they were all

* Observations carried out on behalf of the Royal Medical Research Committee.

curious concerning the recording apparatus and exhibited more interest in it than in the subsequent events. Respiration was unchanged on the average by either the flame or the pistol shot (curves A, Fig. 1 and 2). The pulse rate was increased on the average by one beat per minute after the flame and decreased by the same amount after the explosion (Fig. 3 and 4, curves A). The maximum individual variations were two per minute for both pulse and respirations. Therefore it may be said that there was no appreciable change of the respiratory or pulse rate in normal men.

Response of patients. The response of the patients was in all cases greater than in the controls, but there was a great variation between individuals.

Respiration. The influence of a pistol shot on the respiratory rate was inconstant in character and varied in degree. In three cases there was a slowing by two respirations per minute during the first half-minute, after which the normal rate was resumed. In the remaining nine cases there was an increase in the respiratory rate varying from two to thirteen per minute. The influence of the flame upon respiratory rate was also irregular. Eight cases showed an increase varying between one and twenty-four respirations per minute, while four cases had a decrease of between two to ten per minute.

A comparison of the respiratory response to these two kinds of stimulation in five cases shows a great variation, not only in the relation of case to case, but also in the same case under the different conditions. (Table I.)

TABLE I.

No.	Name.	Change of pulse rate after flame.	Change of pulse rate after pistol shot.	Change of resp. rate after flame.	Change of resp. rate after pistol shot.
1.	Mo.	30+	14+	24+	8+
2.	Ma.	23+	13+	10—	13+
3.	C.	10+	10+	2+	3+
4.	S.	8+	5+	2+	2—
5.	Mor.	3+	5+	3+	8+

In Fig. 1 and 2 the average respiratory rates are shown of various groups of cases. Curve A is in each case that of the controls. Curves B is the average respiratory rate of patients who were fit to return to duty. It will be seen that the variation from the normal is unimportant. These cases closely resemble the normal in their general condition, and this was further emphasised by their mild reaction to emotion. Cases which were of varying degrees of unfitness are averaged in curves C. In curve C of the

TABLE II.
FLAME.

No.	Name.	On-set of symptoms dating from	Exercise pro- ducing material symptoms.	Symptoms occurring on training.	Change of pulse rate.	Change of respiratory rate.
1.	A.	Strain	C.15	X	45+	5+
2.	Mo.	Increased by shell shock	B.15	slight	30+	24+
3.	Ma.	Unknown	B.15	X	23+	10—
4.	C.	Unknown	A.15	X	19+	2+
5.	E.	Dysentery	A.15	—	18+	1+
6.	P.	Gassed	A.B.30	—	12+	2+
7.	S.	Unknown	D.30	very slight	10+	2—
8.	No.	Unknown	D.30	—	10+	1+
9.	Sp.	Unknown	C.30	very slight	8+	2+
10.	Mar.	Unknown	C.30	very slight	7+	10—
11.	Po.	Shell shock	C.30	—	5+	8—
12.	Mor.	Shell shock	C.30	—	3+	3+

TABLE III.
PISTOL SHOT.

No.	Name.	Onset of symptoms dating from	Exercise pro- ducing material Symptoms.	Symptoms occurring on training	Change of pulse rate.	Change of respiratory rate.
1.	E.	Unknown	B.15	X.	22+	6+
2.	Nn.	Shell shock	D.30	—	21+	8+
3.	B.	Unknown	C.15	X	18+	5+
4.	W.	Strain	B.15	—	18+	4+
5.	Mo.	Increased by shell shock	B.15	slight	14+	8+
6.	L.	Unknown	C.15	X	14+	5+
7.	Ma.	Unknown	B.15	X	13+	13+
8.	G.	Enteric fever		—	12+	4+
9.	Sp.	Unknown	C.30	very slight	5+	2—
10.	Mor.	Shell shock	C.30	—	5+	8+
11.	D.	Unknown	D.30	very slight	5+	4—
12.	Gi.	Influenza	D.30	—	2+	2—

flame series the irregularity is caused by the great variation in the response, some showing a conspicuous increase, others a conspicuous decrease in the respiratory rate. The explanation of this is possibly that respiration is partly a voluntary act, and changes in it are on that account less spontaneous. It was found in general that the more unfit the case might be the more likelihood there was of a considerable variation in the respiratory rate after stimulation. The change was not necessarily an increase in the respiratory rate, but might be equally a slowing.

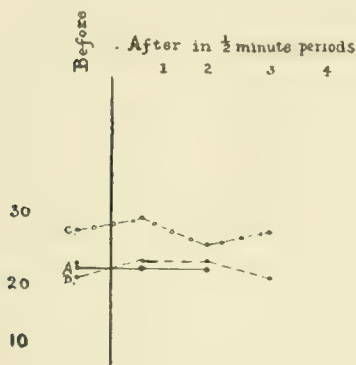


Fig. 1.

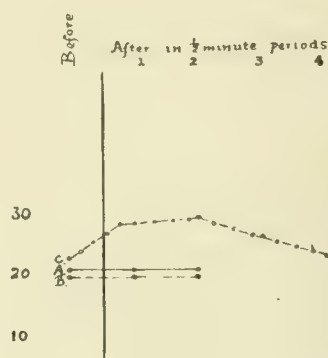


Fig. 2.

- Fig. 1. Curve A. Average respiratory rate of six normal men before and after stimulation by flame.
 Curve B. The same in six cases of so-called "irritable heart" who returned to full duty.
 Curve C. The same in six cases of so-called "irritable heart" who were unfit to return to duty.
- Fig. 2. Curve A. Average respiration rate of six normal men before and after stimulation by pistol shot.
 Curve B. The same in five cases of so-called "irritable heart" who returned to full duty.
 Curve C. The same in seven cases of so-called "irritable heart" who were unfit to return to duty.

Pulse rate. The change of pulse rate after stimulation by the two methods was more constant and exceeded the reaction in the controls in greater degree than is the case of the respiratory rate.

As with the respiration the change from normal was greater as a rule after the flame than after the pistol shot. The general character of the change was fairly constant and differed in different cases purely in the degree of the response. In all cases there was an increase of pulse rate which varied from three to forty-five beats per minute after the flame (Table II), and from two to twenty-two beats per minute after the pistol shot (Table III).

In five cases in which both responses were tested there was a relatively close relationship between the reactions. Those in whom there was a great increase after the flame had a relatively great response after the pistol shot. Likewise, those with a slight reaction to one form of stimulation showed an equally slight reaction to the other.

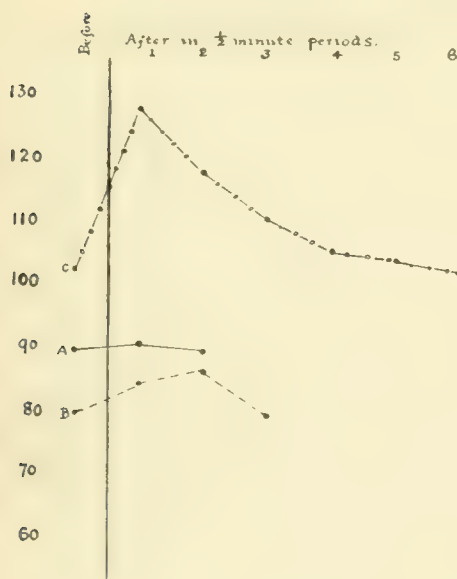


Fig. 3.



Fig. 4.

- Fig. 3. Curve A. Average pulse rate of six normal men before and after stimulation by flame.
 Curve B. The same in six cases of so-called "irritable heart" who returned to full duty.
 Curve C. The same in six cases of so-called "irritable heart" who were unfit to return to duty.
- Fig. 4. Curve A. Average pulse rate of six normal men before and after stimulation by pistol shot.
 Curve B. The same in five cases of so-called "irritable heart" who returned to full duty.
 Curve C. The same in seven cases of so-called "irritable heart" who were unfit to return to duty.

The relation of pulse rate to duration of symptoms. It was deemed of interest to determine what connection, if any, exists between the degree of increase of pulse rate and the duration of the general symptoms. As many of the cases had not been troubled, and therefore did not realise that there was anything amiss until beginning their training as soldiers, the

occurrence and degree of symptoms during this period of their military careers was taken as an index. These observations are tabulated in Tables II and III, and a close relationship is apparent between the degree of incapacity during the period of training and the response of the pulse rate to the stimulations employed. It is not invariable; there are two exceptions, namely, numbers 2 and 4 in Table III, but apart from these cases it appears that the pulse rate in those who have the most marked symptoms during training exhibits the greatest reaction to sensory stimulation.

It was considered possible or probable that those cases which had suffered from shell shock might yield a greater response than those which had not. But no such relationship could be traced.

Relation of pulse response to capacity for work. The relationship between the degree of increase of pulse rate after stimulation and the capacity for work was determined by means of graduated exercises. In the case of the stimulation by the flame, those with an increase of pulse over twelve per minute were unable to tolerate anything beyond fifteen-minute exercises without experiencing material symptoms; while in those cases in which the pulse accelerated by twelve or less beats per minute, longer and more strenuous exercises were accomplished with little difficulty. In the case of the pistol shot the results were less definite. *CASE 2* (Table III) was able to accomplish the most advanced exercises without difficulty, yet showed a very conspicuous increase in the pulse rate when stimulated. Otherwise this series behaves in like manner to those exposed to the flame test.

OBSERVATIONS UPON FAINTING ATTACKS DUE TO INHIBITORY CARDIAC IMPULSES.*

BY THOMAS F. COTTON AND THOMAS LEWIS.

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SOME ten years ago Sir William Gowers published a short treatise entitled "The Borderland of Epilepsy,"¹ in which he described fainting attacks in patients who presented no signs of structural disease, and in whom he noticed attacks of faintness associated with symptoms which he ascribed to over-action of the vagus nerves. Symptoms such as feelings of disquietude in the abdomen, nausea, difficulty in breathing, cardiac sensations, and vasomotor derangements, were described, and Gowers on these grounds referred fainting attacks to over-action of the inhibitory nerves upon the heart. The evidence which he brings forward in his book can hardly be regarded as convincing, though it may be suggestive; for he gives us no observations upon the heart or the pulse which point to his conclusion. His account of the condition which he describes is more philosophic than demonstrative, and it leaves the reader with but a hazy notion of its features and meaning.

Both before and since Gowers' publication a number of observations of a more direct kind have been published which implicate the vagus nerve or its centre as a cause of syncope in man. An account of the more significant of these observations will be found in "The Mechanism of the Heart Beat," published by one of us some six years ago.³ Of recorded instances of syncope of vagal origin a few stand out as splendid illustrations. There is the instance of the man who was attacked by fainting while at stool, and in whom a small aneurism was afterwards found upon the basilar artery.⁴ An expansion of this sac engendered by a rise of blood pressure, so it is supposed, disturbed the medullary centre and produced that slowing of the pulse which was observed and recorded, and its consequent cerebral effects. Another and even more remarkable illustration is the case recorded by Laslett—remarkable because of the thoroughness with which the patient was investigated.² A female patient, in whom no clear signs of structural disease could be found, manifested frequent standstill of the whole heart of four to eight seconds duration which was associated with syncope. In this instance the responsibility of the vagus was proved, for atropine prevented recurrence of the attacks. These two examples may suffice, for although these and others which we might quote implicate the vagus, yet in type

* Undertaken on behalf of the Royal Medical Research Committee.

each of these cases differs essentially from those which it is our present purpose to describe.

It appears to us somewhat curious that accurate observations upon the cardiovascular system during the faints of which young men and women of nervous disposition are the subjects have not as yet been obtained, or obtained, have not been recorded. It is customarily assumed that these attacks are of vasomotor origin; such may be the case, but our present observations throw some doubt upon this explanation and it seems to us desirable that the faints of these young people should be further examined.*

At present we propose to record observations upon a number of young soldiers who are affected with the malady commonly termed "irritable heart." It is a condition in which the response of the heart rate and of the blood pressure to exercise is excessive, and one in which exaggerated breathlessness, palpitation, precordial pain, giddiness and fatigue are associated with exercise. Our opportunities of obtaining data relating to faints, a not uncommon symptom in these patients, have been unusual, since for fifteen months we have been dealing with these men for many hours each day. It has happened on a number of occasions that a man has fainted in the examination room, expectedly or unexpectedly, and that the chance to make complete or incomplete observations upon him has come. Such of the observations as we consider to be of value we relate in the accompanying case reports.

D., aged 24, was a mechanic in the Royal Flying Corps. He had suffered from gastric disturbances, with pain, vomiting and constipation for some years; his work was that of an engineer's fitter, and he played football and went in for cricket and running without trouble before enlisting. He joined in March, 1915, and trained for nine months without symptoms. He was perfectly fit at the end of training and was then transferred to the Flying Corps. After joining the Flying Corps he was vaccinated and was laid up with fever for a week. From this time he became fatigued in his duties. He commenced work in the shops early in January, 1916, and carried out his work without complaint till March, when he went to hospital for two weeks with tonsillitis. He returned to work for seven months and then reported sick with abdominal cramp. He was in hospital a week and was then placed on light duty. A month later he fainted while walking, and again after a fortnight during a brief attack of "influenza." Since this time fainting has been frequent, sometimes occurring twice a day. The attacks come on while he is sitting or standing; the sensation at the onset is that experienced on descending rapidly in a lift. Sometimes fainting comes without warning. Nausea is never experienced. Headaches and exhaustion follow the attacks. Breathlessness, pain over the precordium and palpitation are not experienced.

* One of us (T.F.C.) has recently observed fainting attacks in two young civilians who had been standing in a concert hall for some while. In both instances there were the usual pallor and sweating, and the pulse was impalpable: the first heart rates counted were between 40 and 50.

A poorly nourished, slight man, he has cold hands and is pale; the thyroid is not enlarged. The throat and teeth are normal. The lungs are healthy. The heart is not enlarged; its sounds are normal; the apex beat is localised and strong in the fifth space. The pulse rate while he stands is 60, the blood pressure is 110 mm. Hg.. Mild exercises are not well tolerated; on one occasion he fainted upon exercise; the chief complaints on exercise are giddiness and pain in the chest. The pulse rate is not easily raised by exercise.

February the 12th, 1917. While a sample of blood was being drawn from a vein of the arm, the patient sitting, he complained of feeling unwell. The needle was at once withdrawn. He was pale and was laid back in a backed chair. The pulse was imperceptible. In a moment there was a general tonic straightening of the whole body; consciousness was lost. Immediately after the momentary rigidity vanished the heart rate was counted at 33 per minute at the apex beat; the heart's action was rendered irregular by long pauses. A little later the heart rate was 30; pallor was intense, beads of sweat had appeared on the upper lip; the pulse was still imperceptible. He continued in this state for several minutes. During the gradual recovery and when the heart rate was 60 per minute a curve was obtained from the jugular vein. This curve shows no A-V block, but a long pause affecting the whole heart interrupts an otherwise regular rhythm. This long cycle is somewhat less than the length of two adjacent cycles and resembles the irregularity termed "sino-auricular" block. Five minutes after the onset the pulse was 60 and the systolic blood pressure was 102 mm. Hg.. Some pallor was present still, but consciousness had fully returned.

On further recovery the patient stated that he felt faint and queer at the onset and was nauseated. He had no memory of being moved from chair to chair. He was shaky for some while after the attack.

The same patient was observed in a separate attack on January the 26th. He had been standing for some while waiting examination when he fell suddenly and hit his head on the floor. He lay on the floor breathing heavily and his face was *flushed*. Whether consciousness was lost is uncertain, but he subsequently had no recollection of the armlet being put in place. The blood pressure, one or two minutes after the onset and while the face was flushed, was 130 mm. and the pulse 120; later the blood pressure was unchanged and the pulse had fallen to 92.

The attacks left this patient exhausted and suffering from headache.

March the 2nd, 1917. Preparations were made to draw a sample of blood, and for the introduction of atropine into a vein. A tambour was attached to the wrist. Preliminary records showed the pulse rate to be 115, 105, 102; a needle was run into a vein and a few c.c. of blood drawn; the needle was removed from the arm and the arterial record continued. A few seconds afterwards the pulse rate began to fall, and with the fall the patient became pale and giddy. In 25 seconds the pulse rate had fallen gradually to 63; in another 10 seconds it had fallen to 45; at this point the patient collapsed and appeared to lose consciousness; the pulse could

no longer be recorded. Twenty-two seconds after the collapse one-thirtieth of a grain of atropine sulphate was injected into a vein. Recovery was prompt ; within 25 seconds the pulse was beating at 78 per minute ; within 35 seconds it was 117, the patient had recovered consciousness but was still pale. The pulse rate rose further within the next minute to 143 and continued to beat between 140 and 150 for the remainder of the period of observation. Blood pressure observations were not possible until the stage of recovery, the first reading of 108 mm. Hg. being obtained when the pulse rate was over 140. During the next few minutes it rose to between 115 and 120. There was no sweating during this fainting attack ; the patient experienced nausea at its onset.

F., aged 21, a private in the Durham Light Infantry, was delicate as a boy ; he played no games at school on account of breathlessness and exhaustion. He was never giddy and had not fainted. After leaving school he worked first as an errand boy, later as a painter, and finally on a light job in a brewery. He played football occasionally, but was too short of breath to last out the game ; afterwards he experienced exhaustion. He had never been laid up with any serious illness. He enlisted in September, 1915, and was training for six months, falling out on route marches because of breathlessness, palpitation and fatigue. On two occasions he fell out on drill. He went to France in July, 1916, and was three months on light duty at a base ; he fainted once while on this work while saluting on guard duty ; his symptoms were then similar to those experienced in the attacks which were subsequently witnessed. Later he proceeded to the trenches, where he remained two days ; on marching back he was so distressed that he was sent to hospital and later invalided to England. He was admitted on October the 13th, 1916.

An undersized, poorly developed man of good colour, the hands are warm, moist and tremulous and the axillæ sweating ; the thyroid is not enlarged ; the teeth are in good condition ; the tonsils are not enlarged. The lungs are clear. The heart is not enlarged ; pulsation is slight in the 5th space inside the nipple line. The heart sounds are normal except for a short soft systolic murmur at the pulmonary cartilage. The blood pressure is 112 mm. Hg. ; the pulse rate lying is 84 and standing 80. He was eventually able to do high grade exercises with little difficulty and with little acceleration of the heart's action. He returned to full duty.

December the 18th, 1916. The patient was sitting, and a few c.c. of blood had just been withdrawn from a vein in the arm and the needle had been removed. He began to feel queer, as though his " stomach had turned upside down ;" he became dizzy ; pallor was noticed ; his head fell forward to his knees. He was at once placed in a long easy chair and further observed. By this time the pallor was intense and he was restless. The pulse was imperceptible, the heart sounds were distant, the rate of beating being 50 per minute ; the action was for the most part regular, a single premature beat being noted. From time to time there were retching movements ; the

pupils were little, if at all, dilated; he was limp, mentally confused or actually unconscious for several minutes. A heavy sweat broke out over the forehead and spread over the chest and body; the pallor remained extreme; respiration was slow and sighing. The pulse was imperceptible for several minutes; as it returned the systolic blood pressure was registered (palpatory and auscultatory) at 60 mm. Hg.. A little later the pressure fell to 55 and then to 50, the pulse varying in rate between 50 and 60. Five minutes after the onset some recovery was noted, the pulse had risen to 64 and the blood pressure to 80. Nine minutes after the onset he was able to respond to questions (pulse-rate 88, blood pressure 105). The blood pressure gradually rose to 110 mm. half an hour after the onset, and the man was able to leave the chair and walk supported across the room. The average blood pressure of this man in ordinary circumstances was 118, the pulse rate 80 to 90. He was shaky and exhausted for 36 hours subsequently.

J., aged 28, a private in the London Regiment. He was a clerk for fourteen years before joining the Army and was in good health. He had no previous illness. He enlisted on November the 9th, 1915, and had four months training in England; there was considerable difficulty with the drill and route marches, as he complained of breathlessness and palpitation on exertion. He was never giddy and never fainted. Occasionally he had pain in the region of the heart, headaches were frequent and he sweated very easily. He was given clerical work, but the symptoms persisted, and he was admitted to hospital on September the 13th, 1916.

He is well nourished and has a good colour. He has a few carious teeth with no pyorrhœa, and his tonsils are not enlarged. His hands and feet are clammy; there is no thyroid enlargement. The lungs are clear. There is a diffuse cardiac impulse; the heart is not enlarged; there is systolic murmur at the pulmonary cartilage and there are frequent premature contractions. The pulse rate is 96 and the systolic blood pressure 144. He is unable to do mild exercises without considerable distress; on one occasion while exercising he fainted. The pulse rate is easily raised to a high point by exercise. He went out of hospital to sedentary employment in the service.

October the 3rd, 1916. While standing waiting examination he was noticed to be swaying and breathing uneasily. He was very pale and sweating. He was placed in a chair, but the symptoms continued, although he did not lose consciousness. The first heart rate obtained was 44; the pulse was imperceptible in the initial stage. About two minutes were lost before blood pressure readings could be obtained. The first reading was 124 mm. Hg., the pulse by this time having risen to 90. A little later the blood pressure fell to 112, the pulse increasing to 106. Recovery was practically complete at the end of seven minutes, when the blood pressure was 126 and the pulse rate 116.

B., aged 26, was a private in the Royal Fusiliers. He had rheumatism and bronchitis in 1912. He was a general labourer before joining and always felt strong except for frequent headaches. He was able to go through his

training without difficulty, with the exception of an occasion when he fainted on parade. He complained of breathlessness on exertion, palpitation and precordial pain and giddiness after two months in the trenches in France. A month later he was invalided to England with frost-bitten feet. Two months later, on returning to his regiment, his early symptoms increased. Giddiness at this time was his chief complaint, coming on after exertion or following sudden change from a sitting to an upright position. He never fainted, although he often felt that he would fall. Sometimes when in bed he had a sensation of sinking through the bed. In addition headaches were frequent, sweating was conspicuous, especially of the hands and feet, and there was great lassitude. He was given light duty and was able to carry on for a year with difficulty. With all these symptoms persisting he was admitted to this hospital on February the 10th, 1916.

He is poorly nourished, undersized, pale and sallow. His hands and feet are clammy and his axillæ wet with perspiration. There is a coarse tremor of the hands, the thyroid is enlarged and the pupils dilated. The temperature ranges between 98 and 99.4. The tonsils are not enlarged and the teeth are in fairly good condition. The lungs are clear. The heart is not enlarged and there are no murmurs. The cardiac impulse is diffuse and forcible, the pulse rate when lying is 74 and when standing 96; the systolic blood pressure in the lying position is 154, and in the upright 147.

He is unable to tolerate anything but mild exercises on account of palpitation, giddiness, breathlessness, and exhaustion subsequently. The pulse rate is easily and greatly accelerated by exercise. He was subsequently discharged as permanently unfit for duty.

April the 11th, 1916. While a few c.c. of blood were being removed from a vein in the arm, the patient meanwhile sitting, he collapsed. He dropped forward, there were some convulsive movements of the trunk and upper extremities. He lost consciousness; the pupils were dilated but there was no strabismus. The face was extremely pale and the pulse rate 48 per minute. He was laid flat on the floor. The first blood pressure reading was taken at 100 mm. Hg.; it gradually rose to 116 and the pulse to 60 during the next three minutes. The venous curve at this time was normal.

April the 19th, 1916. Up to the time when blood was taken as a sample the pulse rate was 95 or thereabout. One minute after the removal of the needle from the arm the patient became pale and his head fell forward. For about a minute the pulse could not be felt; it then beat regularly at 45 per minute for several minutes, gradually increasing to 60 while he was under observation. The volume and tension then seemed fair. It is uncertain whether the patient lost consciousness.

McG., aged 20, was a private in the Scots Guards; he had always been in good health, played games and was active in his occupation as a grocer's assistant. He had whooping cough and diphtheria when fourteen years of age, and a sore throat every winter for several years. He enlisted on November the 3rd, 1915, and was in training until March, 1916. He was

inoculated twice, and on each occasion fainted. In March he had scarlet fever, and was in hospital until June. Towards the end of June he continued his drills and then for the first time complained of shortness of breath, pain in the chest and giddiness. Sometimes the giddiness was felt in bed at night. He experienced considerable sweating on exertion, and frequent headaches. On July the 31st, 1916, he was admitted to this hospital.

He is well nourished, the mucous membranes are pale, the hands moist and tremulous. The right tonsil is enlarged; there is considerable pyorrhœa, and there are many carious teeth. The lungs are clear. The heart is not enlarged, and there are no murmurs; the systolic blood pressure is 126 and the pulse rate 72. He is only able to tolerate the mild exercises, on account of breathlessness, precordial pain, palpitation and exhaustion. The pulse rate easily becomes accelerated on exercise. He subsequently returned to light duty in the Army.

August the 2nd, 1916. This patient had been standing for a few minutes and was being examined in this posture when he became pale and said that he thought he was going to faint. Beads of perspiration were visible on the forehead. He continued standing, but swayed and almost fell. The pulse rate was then 45. He lay down and within a few minutes of the onset a normal venous curve, accompanying a ventricular rate of 50 per minute, was taken. The systolic blood pressure was then 100 mm. Hg.. During the attack the patient experienced nausea. Recovery was gradual; ten minutes from the onset the pulse was 72 and the systolic blood pressure 126.

September the 20th, 1916. A sample of blood had been drawn from a vein at the elbow; a few minutes later, while sitting in a chair, he fainted. Pallor was extreme and he lay back limp and sweating with the eyes closed. Observations were delayed for 2-3 minutes. The pulse rate then ranged between 76 and 84, the beats were very feeble, at times imperceptible; the blood pressure could not be measured when it fell below 100; for several minutes it rose no higher than 108. Later and on recovery the pulse rate was 76 and the blood pressure 126. He was then tested as to his reaction to posture. The average pressure standing was 136, and sitting 127.

L., aged 22, was a private in the Gloucesters. He was quite well until five years before admission, when he had rheumatic fever. Since this illness he had been breathless on exertion and had had palpitation. Two years later (1914), while sitting in a theatre, he complained of giddiness for the first time. On three other occasions before joining the Army he had been giddy, but had never fainted. In the past year he has complained a good deal of pain in the region of the heart, usually experienced after exertion. Before joining he was a farm labourer and found the work very difficult because of these symptoms. He enlisted in July, 1915, and had five months training in England. He never fell out when on a route march although he was breathless and had pain in the precordial region; on two occasions he was giddy. He went to the Dardanelles, but soon after his arrival there was invalided to England with joint swellings. Since his return he has had

frequent attacks of giddiness and has fainted twice, on one occasion when getting up in the morning. With the symptoms breathlessness, precordial pain and giddiness, he was admitted to this hospital on February the 7th, 1916. Since his admission he has complained a good deal of nausea and has frequently vomited.

He is well nourished, has slow cerebation; the complexion is sallow, dark rings show round the eyes; the feet and hands are clammy. His teeth are in good condition; the tonsils are not enlarged. Beads of sweat are often to be seen on the face. The hands are tremulous. There is some fullness of the neck, but no enlargement of the thyroid can be distinguished. His temperature varies between 98 and 99. There are no adventitious signs in the lungs; the heart is not enlarged, there is a faint systolic murmur at the aortic cartilage. His pulse rate lying is 55 and standing 81; his systolic blood pressure is 120 lying and 117 standing. His urine is clear; his knee jerks active. He is unable to tolerate anything but mild exercises on account of breathlessness, palpitation, precordial pain and fatigue. He was subsequently discharged as permanently unfit for service.

May the 3rd, 1916. A tambour was attached to the wrist of the patient and a radial curve taken while a few c.c. of blood were drawn from a vein for culture purposes. Three minutes after removal of the syringe he began to turn pale and complained of a sensation of warmth. Beads of sweat appeared on the forehead, nose and cheeks. Sweating and pallor increased and he complained of giddiness and swimminess in the head. He was clearly on the verge of fainting, but it is believed that consciousness was not actually lost. Respirations were sighing in character. The pulse rates are tabulated. During his recovery he was asked to stand and he did so; his symptoms became accentuated and he leaned forward and evidently wished to lie down.

Pulse rate.	
87	Average rate before blood was drawn.
81	During withdrawal of sample.
82	$\frac{1}{2}$ minute later.
85	1 minute.
78	$1\frac{1}{2}$ minutes.
81	2 minutes.
72	$2\frac{1}{2}$ minutes
56	3 minutes (feeling hot and giddy).
72	$3\frac{1}{2}$ -4 $\frac{1}{2}$ minutes (better).
57	5 minutes
50	$5\frac{1}{2}$ minutes
59	6 minutes
60	$6\frac{1}{2}$ minutes
72	7 minutes (better again).
65	$7\frac{1}{2}$ minutes
54	8 minutes
55	$8\frac{1}{2}$ minutes
57	9 minutes
60	$9\frac{1}{2}$ minutes
55	10 minutes
55	$10\frac{1}{2}$ minutes
52	11 minutes
56	$11\frac{1}{2}$ minutes
62	$12\frac{1}{2}$ minutes.

S. This patient was seen on May the 30th, 1916, 1-2 minutes after the withdrawal of blood from a vein for a complement fixation test. He was then intensely pale, and yawned frequently. There was no apparent sweating of the face, but the hands were cold and wet. For about five minutes the pulse rate was 40 per minute, and during this period the systolic blood pressure was read at 70 mm. Hg.. He did not lose consciousness. Recovery was gradual, the pulse rate returning to 60: a venous curve taken at this time was normal.

L., aged 21, was a private in the Scots Guards. From the age of 12 he complained of palpitation, pain in the region of the heart, and giddiness. He had headaches for many years, and a constant feeling of lassitude. He had tonsillitis at 11 and measles at 12. The attacks of giddiness might not come for months, or he might have several attacks in a week. Each lasted a few seconds or a few minutes. He once fainted and fell after squeezing his fingers under a big stone. He once fell while gardening; there was no warning, but he found himself on the ground. Attacks come especially when after sitting for a while he stands suddenly. He is in the habit of steadying himself if he stands from a chair by keeping hold of the chair. He also describes a form of attack which is slightly different and which may come on in any position, even when he lies in bed. This type is accompanied by considerable breathlessness and flushing of the face in patches. It lasts 15-20 minutes and recurs as frequently as the giddy attacks. In them there is never the slightest giddiness but an indescribable discomfort in the pit of the stomach; these attacks frighten him.

He had been a sailor since the age of 13, and joined the Army on February the 18th, 1914, and was seven months in training in England. He went through his training quite easily. He went to France in September, 1914, and was in the trenches until December, 1914, when he was invalided to England with shrapnel wounds in the shoulder, wrist and chest. He returned to France feeling quite fit in April, 1915, and remained on full duty until September, 1915, when he was invalided to England with a wound in the leg. He rejoined his regiment in January, 1916, and then complained of pain in the chest and a return of giddiness and some breathlessness. These symptoms were noticed for several months but were never severe enough to report to the Medical Officer. They gradually increased in severity and he was only able to carry on his duties with difficulty. In April, 1916, he was invalided to England after burial in a mine explosion. Breathlessness on exertion, palpitation, pain in the chest and giddiness were increased; in May he returned to his unit and was given full duty. The symptoms persisted, though with less severity, until November, 1916, when he was admitted to this hospital.

He is fairly well developed, rather pale, with clammy hands and sweating axillæ. He has no thyroid enlargement; there is slight pyorrhœa; the tonsils are not enlarged. There are no adventitious sounds in the lungs. The heart is normal in size and there are no murmurs. He eventually tolerated strenuous exercises without difficulty and was discharged to full duty.

November the 9th, 1916. While this patient was being examined, and after he had been standing for 2-3 minutes, he commenced to breathe deeply and to sigh; he was pale, and when questioned declared himself dizzy and expecting to fall. He yawned continuously while observations were being made. There was no sweating, nor dilatation of the pupils. As he stood the pulse rate varied between 72 and 60, the blood pressure between 80 and 68 (declining); the latter readings were associated with so much giddiness that a chair was placed for him. He sat down. The pulse rate increased to an average of 68, the blood pressure to an average of 98, and during the eight minutes of sitting he felt much better. He then stood again and the following readings were taken.

Minutes after onset.	Pulse rate.	S.B.P.	
14	76	104*	
16	92	100	
18	92	100	
20	88	94	
22	92	100	
24	—	90	giddy
26	88	80	
31	80	82	giddy, yawning, and wanting to sit.
32	88	78	
34	84	86	better
37	84	88	
39	—	86	yawning
41	68	90	sitting and feeling quite well.
44	64	96	
46	64	96	

* Taken by palpation, low readings being confirmed by auscultation.

The patient subsequently spoke of this attack as being of the mild type.

Summary and Remarks.

Fainting attacks, as they occur in soldiers who suffer from "irritable heart," vary much both in frequency and severity. A history of fainting is commonly to be obtained, but those cases are relatively rare in which attacks are repeated at frequent intervals.

As a rule there is warning of the coming attack, or more strictly, there are premonitory symptoms which warn the subject of an impending loss of consciousness; these premonitory symptoms form an integral part of the attack and many of them are attributable to the same cause as the actual loss of consciousness, namely, deficient blood supply to the cerebrum. The early manifestations are dizziness of vision, a sense of uncertainty, or actual giddiness, unpleasant sensations referred to the precordium or epigastrium, and not infrequently nausea; actual vomiting before or during the attack is rare. As a rule, therefore, there is timely warning, but in some instances warning may be brief or absent, and a heavy fall may result when the patient is taken unawares and while standing. Consciousness is lost for a few seconds or for a few minutes. At first there is, as a rule, some general muscular rigidity, though it is uncertain whether this may not be in part volitional.

In some cases this is succeeded by spasmodic movements, confined in the main to the head or upper extremities. Eventually the body is flaccid, signalling the stage of recovery. Recovery in our experience is invariable and no serious ill effects follow; lassitude, headache, the passage of considerable quantities of light urine are the chief after effects and these pass off during the course of the same day or the day succeeding the attack.

In many instances the attack is brought about by a distinguishable cause; the sight of blood, rather than the prick of the needle, is usually responsible when the accident follows the withdrawal of blood from the arm. The receipt of an unusual order, an unusual and alarming incident in the firing line, even the excitement of a medical examination, may be provocative. The erect posture unquestionably predisposes to the attacks in many of the susceptible, especially long standing at attention. In other instances the provocative cause is not to be identified. Where the provocation is known, it acts through reflex channels; the stimulus is received through the special senses, but it may not be—usually is not—the stimulus itself, but the emotional idea to which it gives rise which is ultimately responsible.

Of the predisposing causes of the fainting we know little beyond the frequent association of fainting and the condition termed "irritable heart." It is to be presumed, though it has not been shown, that the attacks are of a similar nature when they occur in young and nervous civilians of either sex. In cases which suffer from "irritable heart," giddiness, transient or enduring, is the rule rather than the exception; but such giddiness is not to be regarded universally as a minor manifestation of the attacks which proceed to syncope. In most cases of transient giddiness we have been unable to detect an alteration of pulse rate; in some there has been an increase of pulse rate. In so far as giddiness is concerned, change of posture is the chief exciting cause; in the case of fainting, posture plays a minor rôle. Our conclusions in respect of the mechanism of disturbed consciousness are therefore confined strictly to the major attacks, where consciousness is lost.

The loss of consciousness is clearly to be sought in anæmia of the brain; and this anæmia is provoked by the combination of two factors, lowered heart rate and lowered force of heart beat.

Reduction of heart rate is almost invariable in the attacks which we are describing, though its degree is inconstant. The previous rates are relatively high as compared with those of normal individuals; during the attack the rate may fall to normal levels, or may fall further to 50, 40 or even 30. Such isolated observations upon the cardiac mechanism as we have been able to make have shown the sequence of auricular and ventricular contractions to be normal during the attack, the heart acting slowly, but at the same time regularly. In one instance irregularity was noticed, and proved to be of sinus origin. The heart rate falls gradually, and hand in hand with the fall the symptoms increase in severity. The gradual fall of rate conditions the premonitory symptoms. The previous heart rate is resumed even more gradually; the symptoms pass off with similar slowness.

Reduction of the force of heart beat is probably invariable. A reduction of heart rate acting by itself would produce a rise of systolic blood pressure* ; in our patients, and during the reduced heart rate of the attack, not only is there no rise but an actual fall of systolic pressure. This fall may be considerable, though its full extent cannot be ascertained ; systolic pressure readings of 60 or 70 mm. Hg. may be obtained, but below these ranges readings are usually unobtainable. The pulse is too weak or is imperceptible. The fall of blood pressure is not attributable to fall of ventricular rate ; it is an added phenomenon. In the syncope of heart-block a lowered systolic pressure for those ventricular beats which occur is presumably absent ; for in arterial records of such attacks the level of the pulse beats is maintained at their apices, and the pulse is forcible. The present form of syncope contrasts with this.

The association of lowered rate with lowered force of heart beat points decisively to a disturbance of the inhibitory mechanism. The not infrequent combination of gastric symptoms, nausea or, more rarely, vomiting, with the cardiac disturbance is in full accord with this conclusion. Where the effects of atropine have been tested, prompt recovery of the circulation has been witnessed. The attacks are of vagal origin and may be relieved by atropine.

In some patients the fall of *heart rate* is the more conspicuous ; in other cases the fall of rate is insignificant and the *lowered pressure* forms the chief outward sign of inhibition. In the last cases, however, the actual loss of consciousness is often in doubt. Variations in the effects of vagal storms from patient to patient are to be anticipated, seeing that similar varying effects are witnessed when the vagi are stimulated in animal experiment.†

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* The mean pressure remaining more or less constant.

† In one of our patients (the first case) an attack (that of January the 25th) is described in which neither pulse rate nor blood pressure was reduced. We have witnessed similar attacks in other patients ; they are accompanied by flushing and not by pallor, and in such cases we have often been undecided as to whether consciousness has been lost ; in some such the genuineness of the attack has been suspected. But, although we hold the vagi to be responsible for the customary seizures, we are not convinced that this is the sole cause of loss of consciousness in these people and would suggest that other forms of attack may still remain unexplored.

OBSERVATIONS ON VENOUS AND CAPILLARY PRESSURES,
WITH SPECIAL REFERENCE TO THE "RAYNAUD
PHENOMENA."*

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UNDUE susceptibility to the influence of moderate cold is a marked and frequent feature in the clinical picture presented by individuals suffering from the condition known as "irritable heart." This susceptibility is shown commonly in the hands and feet. Even in summer, if the weather is at all chilly, patients suffering from "irritable heart" will show, in the majority of instances, abnormal changes of colour, such as lividity or pallor in their extremities. The changes may be brought about by other stimuli than moderate cold, such as emotion, physical exercise, and occasionally for no discoverable reason. Nevertheless, the fact remains that the most common stimulus is cold, and a very moderate change of temperature, such as passing from a warm room to a cold passage, or washing the hands in cold water, insufficient to affect normal people, is sufficient to bring about these changes in susceptible individuals.

The slight degrees of change are so common that they can scarcely be regarded as morbid, but the more marked changes are similar to the conditions described by Raynaud¹ under the terms "local syncope," or "dead finger," when the extremity involved becomes temporarily white, cold and deprived of blood, and "local asphyxia" when the hand is blue and livid. It is not suggested here that these patients are in the early stages of Raynaud's disease, but rather that they commonly show the changes which may be described as the "Raynaud Phenomena."

The greatest divergence from normality in the "irritable heart" cases is seen in those patients whose hands are always livid, numb and cold. In these cases the paroxysmal element is lacking, the patients say that their hands always are, and always have been, blue and cold. For this reason, the influence of external changes of temperature is not apparent. The hands may be of a uniform dark blue colour, which usually extends up to the wrist.

* A report to the Medical Research Committee.

Commonly a bluish tinge can be seen on the forearm. The nails are slate coloured. When pressure is exerted on the hand, a dead white patch is produced, which slowly regains its former appearance, the colour spreading uniformly from the periphery of the patch to its centre. If the hand is pricked, a drop of dark blood slowly escapes. These three points, the coldness, the slow return of colour when a white patch has been produced by pressure, and the darkness of the blood, suggest that the capillary circulation has been slowed and a condition of venous stasis produced. In these cases it is uncommon to obtain a history of "dead finger," and there is no complaint of pain or tingling, but rather of numbness and inability to do fine work. Excessive sweating of the hands may be a cause of annoyance. The feet are usually also blue and cold. There is no accompanying cyanosis of the mucous membranes. Occasionally hands are seen which are blue and warm, but these are the exception.

Another type of case shows a marked mottling of the hands. The general colour is blue, but there are patches of pink, with fairly well defined margins, and these patches are constant in position. The hands vary from day to day, and sometimes the patches are scarcely noticeable, but when they are apparent, they are always in the same places. If pressure be made on one of these pink spots and a white patch produced, the same slow return of colour from the periphery inwards is noticed as in the blue hand, but in addition there is often, in the centre of the patch, a pink point, which can only be obliterated by extra and considerable pressure. From this point, if present, after pressure is removed, a wave of colour will spread outwards until it meets the wave of colour spreading inwards from the periphery. Reference will be made to the origin of the pink patches later on in the paper. This type of case usually shows some abnormality of colour even in hot weather.

The most common history met with in these cases is as follows:—The hands are normal in colour in hot weather, but turn blue in slight cold, and the fingers often go dead in the winter. These cases are distinctly paroxysmal in type, and the changes may be brought about by other stimuli than cold. Some patients say that open-air exercise makes their hands more blue, but exposure to the outside air may still be the exciting cause. Others state that the attacks come on quite spontaneously, sometimes while they are sitting quietly by the fire in the evening. Pain is usually absent when the hands turn blue, but tingling is complained of when recovering from an attack of "dead finger." Sweating is very marked in this type of case, more especially in hot weather when the hands are normal in colour. This wetness of the hands in warm weather is often more troublesome than the blueness and numbness in winter. The veins of the back of the hand are usually less prominent when the hand is blue than when it is normal in colour. It may be difficult to follow their course, even if the hand is hanging down. The hands often look slightly swollen, but there is no pitting on pressure. The difficulty in seeing the veins may be partly due to this puffiness, possibly it may be partly due to the contraction of the veins.

It will be obvious from the foregoing remarks that the condition known as "local asphyxia" or "local cyanosis," to use the term suggested by Barlow,² was more common in this series of patients than the condition known as "local syncope" or "dead finger." The frequency and permanence of the condition suggested an investigation of the blood pressure in the veins and capillaries of the extremities involved.

Methods.

In these observations all the measurements of venous and capillary pressures were made by means of the improved Hooker apparatus³ for estimating venous pressure.* The subject is seated by the side of a bench and the hand and forearm rest on the bench, avoiding as far as possible any muscular strain. His seat is arranged so that the veins on the back of the hand are on the same level as the subcostal angle. This point was selected by von Recklinghausen for the heart level, and as most of the recorded observations in venous pressures are given with reference to this heart level, it has been adhered to throughout this series. When the level of the hand is adjusted, a prominent vein on the back of the hand is selected, and the capsule placed over it and held in position by a small rubber band circling the hand. The capsule is then fixed to the skin by a film of celloidin. When this film is quite dry, the rubber band is removed, and the air chamber tested to see if there is any leak. To read the venous pressure, air is forced into the chamber until the vein is seen to collapse. At the point when the pressure inside the chamber, as recorded by the manometer, equals the venous pressure, the vessel will collapse and the shadow of the vein disappear. The most satisfactory readings are obtained by taking the mid-point of pressure when slight oscillations cause the vein shadow to disappear and reappear, as the vessel collapses and fills again. After the venous pressure has been read, the capillary pressure is determined. The pressure inside the chamber is increased until the skin is blanched, and a further rise of pressure produces no more whitening. There is naturally a considerable error in observation, but this can be reduced by taking numerous readings, until fairly consistent results are attained. The manometer supplied with the Hooker apparatus records up to a pressure of 30 centimetres of water, and is intended only for the estimation of venous pressures. A 30 cm. water pressure was found to be insufficient to blanch the skin of the blue-handed patients. A similar apparatus was therefore constructed with a manometer recording up to 50 cm. of water. All the readings were taken between 10 and 12 o'clock in the morning, so that changes due to a possible diurnal variation⁴ were eliminated.

* This apparatus consists of a small capsule 20 mm. in diameter, 10 mm. high, and made of glass about 1 mm. thick. At the side a glass tube, slightly tapered, serves to connect the chamber with a water manometer. The pressure is regulated by squeezing a small rubber bulb, which is connected by a T-opening with the manometer, and the tube leading to the glass chamber.

Results.

The striking feature of the results is the high degree of capillary pressure found in the blue-handed patients. Table I gives the results of a series of seventeen observations on venous and capillary pressures in normal individuals, six men and four women, taken under the same conditions as the observations on the patients.

TABLE I. (CONTROLS.)

Hands normal in colour.

MEN.			WOMEN.		
	Venous pressure in cms. of H ₂ O.	Capillary pressure in cms. of H ₂ O.		Venous pressure in cms. of H ₂ O.	Capillary pressure in cms. of H ₂ O.
L.	10.0	23.0	B.	8.0	22.0
..	9.0	20.0	..	9.0	22.0
..	12.0	24.0	..	8.0	22.0
H.	11.0	26.0	M.	9.0	22.0
D.	10.0	24.0	G.	7.0	22.0
S.	9.0	23.0	..	8.0	19.0
B.	11.0	24.0	..	10.0	22.0
..	12.0	26.0	J.	11.0	27.0
C.	11.0	22.0			
Average	10.5 cms.	23.5 cms.		8.7 cms.	22.2 cms.

In the men the average pressure in the veins on the back of the hand was equivalent to a pressure of 10.5 cms. of water (range 9-12), the average capillary pressure in the same region 23.5 cms. of water (range 20-26). The eight observations on women gave slightly lower readings—average venous pressure 8.7 cms. (range 7-11), average capillary pressure 22.2 cms. (range 19-27). Hooker⁵ has found a lower average venous pressure in women than in men.

In Table II the patients have been divided into three classes; in Class 1 the hands were normal or red in colour, when seen on one or more occasions; in Class 2 the hands were blue or had a bluish tinge, when seen on one or more occasions; in Class 3 the patients were all seen on several occasions,

and the hands were sometimes normal, sometimes blue. It is quite likely that if the patients in Classes 1 and 2 had been seen more frequently, some might have come into Class 3.

TABLE II. (PATIENTS.)

CLASS 1.			CLASS 2.			CLASS 3.				
Hands normal or flushed.			Hands bluish or blue.			Hands sometimes blue, sometimes normal.				
	V. P. in cms. of H ₂ O.	C. P. in cms. of H ₂ O.		V. P. in cms. of H ₂ O.	C. P. in cms. of H ₂ O.		Normal.		Abnormal.	
				V. P.	C. P.		V. P.	C. P.		
Harrop	11.0	26.0	McMurray	—	42.0	Entwhistle	9.0	22.0	—	—
"	12.0	22.0	Everstield	12.0	34.0	"	9.0	21.0	—	—
"	13.0	24.0	Atkins	13.0	—	"	—	—	13.0	32.0
Street	15.0	24.0	"	13.0	40.0	Thompson	10.0	20.0	—	—
"	14.0	28.0	Steer	13.0	26.0	"	—	—	10.0	32.0
Woolf	13.0	24.0	"	12.0	30.0	Dale	9.0	26.0	—	—
"	11.0	33.0	Robertson	15.0	50.0	"	—	—	11.0	35.0
Johnson	17.0	26.0	"	15.0	50.0	Carter	11.0	26.0	—	—
Cook	9.0	20.0	Carlisle	—	34.0	"	—	—	10.0	26.0*
McClean	—	34.0	Hawkins	—	50.0	"	—	—	10.0	46.0†
Yates	10.0	32.0	"	—	46.0	Pace	14.0	26.0	—	—
Lawson	9.0	21.0	"	—	50.0	"	11.0	26.0	—	—
Foster	9.0	22.0	"	—	50.0	"	—	—	11.0	30.0
Edwards	10.0	28.0	Howarth	11.0	34.0	"	—	—	10.0	36.0
"	10.0	28.0	Hooker	—	42.0	Shelford	12.0	27.0	—	—
"	10.0	27.0	McGinnity	—	46.0	"	—	—	13.0	30.0
"	10.0	28.0	Wren	—	42.0	Shuffle	9.0	30.0	—	—
Costigan	10.0	28.0	"	—	46.0	"	12.0	24.0	—	—
Newson	8.0	24.0	"	—	46.0	"	10.0	28.0	—	—
Shearing	10.0	25.0	Robinson	—	30.0	"	—	—	9.0	32.0
Barnard	10.0	26.0	"	—	32.0	Parrott	11.0	26.0	—	—
Boswell	10.0	26.0	Ely	15.0	34.0	"	11.0	28.0	—	—
Neale	12.0	26.0	Ellis	—	34.0	"	—	—	9.0	34.0
Baxter	11.0	28.0	Ellison	9.0	33.0	"	—	—	—	—
Mercer	10.0	28.0	"	11.0	32.0	"	—	—	—	—
Horsman	10.0	26.0	Roberts	10.0	30.0	"	—	—	—	—
"	"	"	"	10.0	24.0	"	—	—	—	—
"	"	"	Duckworth	8.0	30.0	"	—	—	—	—
"	"	"	Hoad	11.0	31.0	"	—	—	—	2
"	"	"	"	11.0	32.0	"	—	—	—	—
"	"	"	"	11.0	34.0	"	—	—	—	—
"	"	"	Lee	9.0	32.0	"	—	—	—	—
"	"	"	Brown	11.0	28.0	"	—	—	—	—
"	"	"	Davis	9.0	33.0	"	—	—	—	—
"	"	"	Stangon	12.0	30.0	"	—	—	—	—
Average	10.9	25.9		11.4	36.9		10.6	25.3	10.6	33.3

* Slightly blue.

† Very blue.

In Class 1 the average venous pressure was 10.9 cms. of water (range 8-17), the average capillary pressure being 25.9 (range 20-34), practically the same readings as those obtained in the male controls. In Class 2 the average venous pressure was 11.4 cms. (range 8-15), the average capillary pressure 36.9 cms. (range 24-50). Some of the slaty-blue hands were not completely blanched even by a pressure of 50 cms. of water. In Class 3, when the hands were normal in colour, the average venous pressure was

10.6 cms. (range 9-14), and average capillary pressure 25.3 cms. (range 20-28) ; when the hands were abnormal in colour the average venous pressure was 10.6 cms. (range 9-13), and the capillary pressure 33.3 cms. (range 36-46).

TABLE III.

Average venous and capillary pressures in controls and patients.

CONTROLS.			PATIENTS.		
	V. P. in cms. of H ₂ O.	C. P. in cms. of H ₂ O.		V. P. in cms. of H ₂ O.	C. P. in cms. of H ₂ O.
Men	10.5	23.5	Class 1 Hands normal	10.9	25.9
			Class 2. Hands blue	11.4	36.9
Women	8.7	22.2	Class 3. Hands variable. When normal	10.6	25.3
			When blue	10.6	33.3

The venous pressure is obviously little affected, only showing a slight rise in the blue-handed patients, while the capillary pressure in such patients is markedly raised. In short, on seeing a blue hand, it is safe to predict, in nine cases out of ten, that an abnormally high capillary pressure (over 30 cms.) will be found, but it is impossible to predict the height of the venous pressure. It will be noticed that the venous pressure is omitted in several cases in Class 2. This is due to the difficulty of obtaining satisfactory readings when the vein is scarcely visible, a condition frequently found in the chronic blue hands.

When the glass chamber was placed over a pink spot in an otherwise blue hand, it was found that the highest pressure (50 cms.) which could be recorded would not blanch the skin completely. Although the greater part of the skin inside the chamber was blanched by a pressure varying between 40 and 50 cms. of water, there remained a central spot or streak of pink which was not blanched when the pressure was raised to the full height of 50 cms.* To compare the capillary pressures in the blue and pink areas, readings were taken in the following manner. Two capsules were fixed to the back of the hand, one on a blue area, and one on a pink area. The capillary pressure in the blue area was determined, and then the pressure was raised to the same height in the chamber over the pink spot. It was found that the

* The celloidin collar to the capsule will not hold with a pressure raised above 50 cms. of water.

skin of the periphery of this area became blanched, but a central pink spot or streak remained, which could not be blanched by a further rise of pressure (up to 50 cms). It seems fair to conclude that a pink spot is due to a dilated arteriole, the general blood pressure from the heart and arteries being transmitted more readily to the capillaries supplied by that vessel, and thus causing a localised area, in which the capillary pressure is abnormally high.

It is well known and easily demonstrated that if one hand is placed in cold water, the veins of the other hand can be seen to contract and to become less prominent. An attempt was made to trace the sequence of events in the capillaries and veins when this reflex response to cold takes place. The experiment was carried out in the following manner. The glass capsule was fixed to one hand, and the venous and capillary pressures at heart level determined. The pressure was then kept steady in the manometer at a point slightly below the venous pressure, so that the vein was still plainly visible. The other hand was then placed in water drawn from the cold tap (17-19° C.). If the vein collapsed, a fall of pressure inside the vessel must have taken place. After a rest and warming the hand, the experiment was repeated, except that the manometer was held so that the pressure in the air chamber was just below the capillary pressure, and a faint pink colour could still be seen in the area. The other hand was again placed in cold water, and if the skin became completely blanched, a fall in capillary pressure must have taken place, while if the skin became pinker, the pressure must have risen. The changes in colour due to alteration in capillary pressure could be followed fairly easily, but the changes in venous pressure with some difficulty. The sequence of events as seen in one patient will be given, as in him both venous and capillary readings were very clear, and experiments with other patients gave similar results.

Sergeant H. had been six years in the Army: he was three years in India, and frequently in hospital with fever, debility, etc. About three months ago, when serving in the trenches in France, he developed symptoms of breathlessness, palpitation and precordial pain. He had always experienced cold hands, which were blue and numb in the winter. Sometimes the four fingers up to the middle joint would become white, numb and cold. A tingling sensation was present when the circulation was restored. In hot weather the hands would sweat very much. The patient was more bothered by the dampness of the hands in hot weather than by the numbness in winter. While recovering from fever in India, he had an attack of amblyopia, which lasted a few minutes; he described the sensation "as if looking into a mist." This is the only patient in which a history of transient "obscuration of sight," such as Raynaud described,⁶ has been obtained.

On the day of the experiment the hands of this patient were rather flushed and the nails pink. There was no blue tinge, except when the hands were held for some length of time in one position.

The venous pressure with the hand at heart level was 13 cms. of H_2O , capillary pressure 24 cms.. The pressure in the manometer was maintained steadily at a pressure of 10 cms., so that the vein on the back of the right hand was plainly visible. The left hand was then placed in cold water. The vein in the right hand slowly collapsed and then reappeared. At the end of about one and a half minutes the vein was as prominent as at the beginning of the experiment. (A control experiment was first carried out, omitting immersion in the cold water.) When the left hand had been rested and warmed, the pressure on the right hand was maintained steadily at a height of 20 cms. of water. The skin inside the glass chamber showed a faint pink colour. When the left hand was placed in cold water, a blanching of the skin inside the glass chamber took place at once (within 5 seconds) and was immediately followed by a return of colour, until the area of skin was pinker than at the beginning of the experiment. Experiments with other patients gave the same sequence of changes, with variations in the time, but usually the blanching took from 5 to 10 seconds, and was followed by a return of colour in another 5 to 10 seconds. At the end of a minute the colour was deeper than at the beginning of the experiment. The same experiment carried out on controls with normal hands and no history of blueness or "dead finger" showed a similar blanching of the skin in the first few seconds, but although the colour returned to nearly the same depth in from 10 to 30 seconds, in no case (four controls) did the tint become pinker than at the beginning of the experiment. In the controls the changes in venous pressure were not very obvious. In response to the stimulus the vein appeared to get less prominent and did not return to its original size. The reflex response to a very moderate stimulus of cold (water at $17-19^{\circ}C.$) appeared to be as follows. In patients whose hands readily become blue in cold weather, the first change was a fall in capillary pressure, as evidenced by the momentary blanching; this was followed in the next few seconds by a return to the normal capillary pressure, and again a few seconds later by a definite rise above the normal. In the veins there was a preliminary fall, followed by a rise of pressure to normal or above. In the controls the same transient fall in capillary pressure was seen, but there was no rise above the normal. The venous pressure fell a little, but no definite subsequent rise could be detected.

Pathology.

The outstanding feature of the blue-handed patients is the capillary stasis. This stasis may be produced in several ways. By contraction of the arterioles: the supply of blood and therefore the *vis a tergo* is diminished—the blood does not circulate so quickly. By contraction of the venules: a greater resistance is offered to the outflow from the capillaries and the capillary area becomes engorged. Capillary stagnation produced by the former mechanism would be accompanied by a low capillary pressure, but if produced by the latter mechanism by a high capillary pressure. In these

cases the capillary pressure is found to be high. The condition is therefore more probably due to increase of resistance in the venules. A third alternative must be considered, namely, a simple dilatation of the capillaries. Such a dilatation would undoubtedly produce a lowering in the velocity of the blood, and therefore a relative deoxygenation. The capillaries would be fuller than normal, with the result that the hand would become flushed, and if the deoxygenation was sufficiently marked, a blue tinge might develop. But a capillary dilatation, in the absence of venous constriction, would hardly explain a rise of the capillary pressure to a point which may be double the normal.

The explanation of "dead finger" and "local asphyxia" offered by Raynaud was as follows:—"It (the paroxysm) commences by a spasm of the capillary vessels . . . In the simplest cases . . . the exaggerated peristaltic contraction of the capillaries drives the blood before it, the extremities become pale, withered looking and insensible. This is "dead finger." But the phenomenon does not persist long enough for gangrene to follow. To contraction succeeds relaxation, the circulation is re-established, and everything returns to the normal state after a period of reaction more or less painful. Such is "local syncope," in which the venules participate in the contraction of the arterioles. "Local asphyxia" is only a more advanced condition. After an initial period of capillary spasm there occurs a period of reaction, but it is incomplete reaction. The vessels which return first to their primary calibre, or even beyond, are naturally those which present in their structure the fewest contractile elements, *viz.*, the venules. At the moment when these are opened, the arterioles being still closed, the venous blood, which had been first driven back into the great trunks of the dark blood system, flows again into the finest vascular divisions, and then the extremities will take on that tint varying from blue to black which is a certain index of the presence of venous blood in the capillary network . . . in other terms, syncope precedes asphyxia."

But in the present series of cases, this order is commonly reversed. "Local asphyxia" is the chronic condition in some cases, the first stage of the paroxysm in others. "Local syncope" is transient and follows the asphyxia in many cases, but in a few there is a record of "dead finger" without any accompanying blueness. Raynaud's theory explains the case of "dead finger" followed by asphyxia, but it seems difficult to explain the condition of "local asphyxia" alone, with a high capillary pressure, except on the theory that the condition is mainly due to an over-contraction of the venules. Taking into consideration the changes in pressure noted when the reflex response to cold is tested, the most probable sequence of changes appears to be as follows:—The first change which takes place both in patients and controls is a transient fall in capillary pressure. This is probably due to a constriction of the arterioles, which diminishes the supply of blood to the capillaries, in which the pressure is therefore reduced. If at the same time the venules contract, the capillary pressure may become equal to normal

again (owing to increased distal resistance), though the flow of blood through the limb be diminished.

That this diminution of flow as a response to cold takes place is proved by the experiments by G. N. Stewart⁷ and Hewlett.⁸ G. N. Stewart showed by means of a calorimeter that if one hand is placed in cold water, the flow of blood through the other hand is cut down to nearly one half. This change of flow occurred at once and was permanent for the duration of the observation (6 mins.). Hewlett's observations with the plethysmograph gave similar results. Stewart also made the interesting observation in two cases of Raynaud's disease, that immersion of the right hand in cold water instantly cut down the flow in the left from 6.67 to 2.97 grams per 100 c.c. per min., a very strong reflex vasomotor effect. This diminution was much more transient than normal, and gave way to an increase even when the right hand continued in water. He suggests that these observations support Raynaud's theory of instability of vasomotor adjustment.

In our controls the capillary pressure does not quite return to normal, but in the patients the capillary pressure rises eventually above the normal. This suggests that the chief vascular spasm is distal to the capillaries. The likelihood that this spasm takes place in the venules is supported by the observation that the larger veins on the back of the hand in "local asphyxia" are often so contracted down as to be almost invisible. If the spasm is continued long enough, the velocity of the blood stream in the capillaries must be diminished, owing to the increased distal resistance. This leads to cooling of the blood, greater loss of oxygen and consequent venosity.

The suggestion has already been put forward that the pink areas in blue hands are due to the dilatation of isolated arterioles. It has been shown by Bayliss⁹ and others that an increase of CO₂ in the blood will cause dilatation and loss of tone of the arterioles and capillaries. It would not be surprising then to find that, although constriction of the arterioles is the first response to cold in susceptible patients, yet this constriction may be followed by dilatation, when the blood in the capillaries and smaller arterioles is sufficiently venous to exert a deleterious effect upon their tone.

The following case illustrates the changes seen in a patient with paroxysmal attacks.

J. P. Joined the Army in 1915 and went to France in March, 1916. He first noticed changes in the hands during the winter of 1916-17 in France. The first and middle fingers of the right hand occasionally went dead. The hands had gone blue at times, but the left never so blue as the right. The hands were cold in the winter, but of ordinary colour in hot weather.

This patient was first seen on June 16th, a very hot day. The hands were normal in colour and sweating. The pressure in the veins on the back of the hands was equivalent to a pressure of 11 cms. of H₂O, and the capillary pressure to 26 cms.. On June 30th, a chilly day, the patient was seen during a paroxysmal attack. He had been waiting for some time in a cold passage,

which had brought on the attack. When first seen, at 10.30 a.m., both hands were blue; the tips and nails of the four fingers of the right hand were a deep slaty blue. The pad of the right forefinger was white and dead-looking. Both hands were numb and cold. After eight minutes the little finger and ring finger were red, the nails pink, the middle finger was red on the outer and blue on the inner side, the nail was partly blue and partly pink. The nail of the forefinger was still slaty blue; the white area on the pad of the forefinger had turned blue. In another eight minutes all the blue tinge had disappeared from the middle finger, but the nail of the index finger was still slaty blue. The little and ring fingers were warm to the touch, but the thumb and forefinger were cold and numb. At this moment the difference in temperature between the outer and inner sides of the hand was very striking. The blue colour gradually disappeared from the forefinger and at the end of another fifteen minutes the nail was completely pink and the pad of the forefinger normal. At 11.5 a.m. the venous and capillary pressures were read. At the moment there was a faint bluish tinge over the whole hand, but the finger-tips were pink. Sensation was normal, except in the thumb, which was still cold and numb. The outer side of the hand was warmer than the inner. Venous pressure 10 cms. of H_2O , capillary pressure 36 cms.. This shows a much higher capillary pressure than the reading (26 cms.) on the day when the hands were normal. The patient was seen again three days later, on a cool day. The hands were red with a bluish tinge, the nails were pink; no paroxysmal attack of "dead finger" had taken place as on the previous date. The venous pressure was equivalent to a pressure of 11 cms., and the capillary pressure to a pressure of 30 cms. of water.

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BRIEF RESUMPTION OF AURICULAR ACTIVITY IN A CASE OF OLD-STANDING AURICULAR FIBRILLATION.

By EDGAR LEA.

(*Manchester.*)

THE following case was under continuous observation for over two years. During this time many graphic and electrocardiographic curves were obtained, on all of which occasions, except the one to be presently noted, the characteristic features of auricular fibrillation were present. On this occasion an electric curve was taken; in the general irregularity of its rhythm it seemed to differ in no way from that customarily present, and it was only upon the development of the plate afterwards that clear evidence was forthcoming that the auricular action had been resumed. Though many subsequent curves were gained a similar event was never again recorded.

Clinical account. Mrs. E., aged 51, came to my out-patient department at the Salford Royal Hospital on August the 3rd, 1913, extremely ill with intense dyspnoea, palpitation, some cough, and dropsy of the legs. The pulse was very rapid, 160, and quite irregular. She was admitted at once.

History. The patient had suffered from shortness of breath since the age of 11. When aged 15 she often suffered from attacks of dizziness, for which, at times, she would have to give up her work. About this time also she had growing pains. She never had either chorea or rheumatic fever, but says that her mother suffered from both these complaints. She married and had nine children. For many years, however, she had suffered from cardiac symptoms. There was shortness of breath on exertion, palpitation for almost as long as she could remember, and on several occasions she had swelling of the legs and stomach. She had been under many doctors and, two years previously, had attended this hospital for some time. Pain in the region of the heart was frequent. Sometimes induced by effort, sometimes arising spontaneously, it was of a gnawing type, and usually limited to the region of the heart, but, on occasion, it would radiate along the outer aspects of both arms. Often the pain would persist for two or

three days, during which she would feel generally poorly. The patient when first seen said she frequently experienced cold hands and feet, that the throat sometimes felt tight, that a hard cough was often present, and that she often had a watery discharge from the right nostril. She sweated very readily.

Condition on admission. August the 3rd, 1913. Patient was a tired and anxious-looking woman, rather cyanotic and obviously distressed with her breathing. She could not lie down. There was marked venous pulsation in the neck. The heart's impulse was weak and diffuse over a large area. The dulness was as follows: third rib above; $1\frac{1}{2}$ inches to right and $6\frac{1}{2}$ inches to left of the mid-sternum respectively. A soft systolic murmur was audible over the apex and at the lower end of the sternum. A short but distinct diastolic murmur was limited to the apex. The heart rate was very rapid, 162, and irregular. There were moist sounds at both bases of the lungs and the percussion note was impaired. There was marked tenderness to pressure over the region of the liver and there was probably free fluid in the abdominal cavity. A trace of albumen was present in the urine.

Whilst being examined she had an attack of faintness and exhaustion and showed increasing distress, for which stimulants were given.

Progress. From August the 4th to 6th the patient was still very ill, the pulse rate had not been much slowed by tinct. digitalis, m. 10 (t.d.s.).

August the 6th. 0.3 c.c. of strophanthone, diluted with an ounce of normal saline, was injected intravenously. The effect was very striking; the subjective symptoms were relieved in half an hour and in the same time the pulse was reduced from 160 to about 90 per minute. The irregularity of rhythm, however, remained. Graphic tracings showed the typical features of auricular fibrillation, both before and after the injection.

August the 9th. Patient much better; less dyspnoea and palpitation, and no pain. Given tinct. digitalis, m. 20 (t.d.s.). Pulse rate, 100, irregular.

August the 18th. Progress uninterrupted; the patient felt and looked better, and there had been considerable diuresis. Digitalis stopped.

August the 25th. Still feeling well, but the pulse rate had gone up to 150 and remained irregular. Tinct. digitalis resumed, m. 5 (t.d.s.).

August the 29th. Still well. Pulse rate, 152. Tinct. digitalis, m. 15 (t.d.s.). This dose was maintained till the patient's discharge from hospital on September the 8th. At that time she said that she was better than she had been for many months. On two occasions during her stay the effect

of atropine was tried: once when she was taking full doses of digitalis (August the 11th), and again when off this drug (August the 25th). When on digitalis the acceleration was 90 to 150, a 66·6% increase: when not on digitalis, acceleration 130 to 150, equal to 13·7%.

November the 15th. Seen again, she was not so well. All the old symptoms had returned with the exception of cedema. She had not been having any medicine. She was placed on digitalin granules, one daily.

November, 1913, to April, 1914. She attended the hospital regularly and continued in good general condition. The pulse on every occasion was irregular, but its rate was readily controlled by digitalis. Occasional tracings confirmed the original findings of auricular fibrillation.

May, 1914, to May, 1915, I lost sight of the case as I had altered my hospital duties. During some of this time, however, she had attended the hospital.

May the 8th, 1915. Again seen, she had had no medicine for several months. She was again extremely ill and in exactly the same condition as when first seen (August, 1913). The pulse was irregular, rate 160, and tracings again showed auricular fibrillation. She was given a hypodermic injection of digitalin, 1·50 gr., at once, and the tinct. digitalis, m. 20, was ordered to be taken four-hourly.

May the 15th. Much improved. Heart rate, 132; pulse rate, 76. Tinct. digitalis, m. 20 (t.d.s.).

May the 22nd. Heart rate, 106; pulse rate, 76; cedema now gone.

May the 29th. Heart rate, 106, respiration, 36; after effort, a walk of about 150 yards, heart rate, 160; respiration, 48. The effect of belladonna was now tried by adding to the digitalis mixture, tinct. belladonna, m. 15.

June the 5th. For the last week the patient had not been quite so well. She had had much palpitation, which had come on even at rest, and at times had wakened her from sleep. The breathing was improved, however, and she took her food better. Heart rate, 116, respiration, 32; after effort, 148 and 42 respectively.

July the 3rd. Belladonna was now stopped and the effect of jaborandi (m. 30, t.d.s.) was tried.

July the 10th. Patient feeling better. Heart rate, 88; after effort, 140.

July the 14th. Still improving. Heart rate, 80. Still irregular. Jaborandi stopped and tinct. belladonna, m. 10 (t.d.s.), given instead.

July the 21st. Patient keeping well. Heart rate had accelerated somewhat and was now 100 (after effort, 128). It was on this occasion, when the patient was under the influence of moderate doses of belladonna, that the resumption of auricular action was noted (Fig. 2).

July the 29th. Patient not so well. Occasionally dizzy. The heart rate was now 160; pulse rate, 84.

From this time onwards till I last saw her in September, 1915, I kept her under digitalis all the time. She remained in good general condition, and the pulse, though irregular, kept within normal limits of speed. I lost sight of her, but subsequently learnt that the patient died in March, 1916.

Fig. 1 shows the electrocardiogram usually obtained in this case. It is typical of auricular fibrillation. There is no sign of any *P* wave, *T* is inverted in lead *III*, upright but not prominent in leads *I* and *II*. In lead *III* a single premature ventricular contraction is seen, of left ventricular or apical type. The rhythm is irregular, the rate 130.

Fig. 2 represents the single observation of resumed auricular action. Like the former curve, the rhythm is quite irregular, the rate 96, and the form of waves *R* and *T* are unchanged. Now, however, we see a distinct *P* wave, preceding, by a normal time interval, each ventricular complex. The *P* wave presents certain changes in character. Usually it is upright and fairly prominent, but sometimes it is inverted. In lead *III*, four premature ventricular contractions appear, of the same type as seen in Fig. 1. Their presence obscures the *P* wave, a fact to be anticipated in the latter portion of the curve, which alone shows a regularity of rhythm.

It is generally and correctly held that auricular fibrillation, when present continuously for years, may be regarded as permanent. The pulse has, indeed, been called the *pulsus irregularis perpetuus*. In such cases the characteristic arrhythmia alone affords strong evidence of this disordered auricular action. But an accidental observation has shown (Fig. 2) that, even in a case of this character, it is possible that the auricles may resume their normal action. It is important to note, too, that such an event may not interrupt in any way the general character of the arrhythmia as felt at the wrist or over the heart. In this case the irregularity of rhythm was as marked with normal auricular activity as when the auricles were fibrillating. It would appear from the record of this patient that fibrillation of the auricles, even after it has persisted for a long while, may disappear, or that in cases where persistent fibrillation may seem to have been present, a normal auricular action may be resumed as a transient event. The frequency with which the last change may happen in a given case or in different cases is quite unknown.

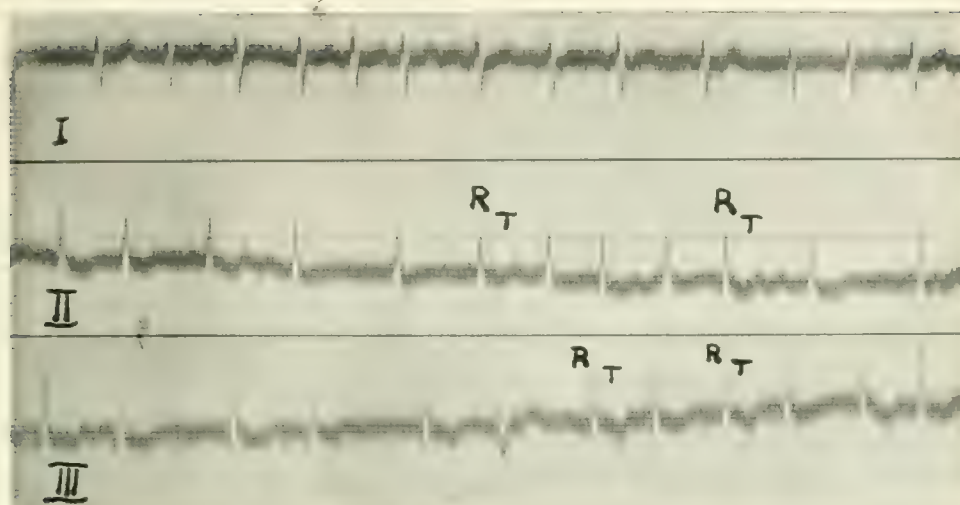


Fig. 1. Electrocardiogram taken August the 15th, 1913, showing typical auricular fibrillation. In lead *III* a single premature ventricular beat of left ventricular or apical type occurs.

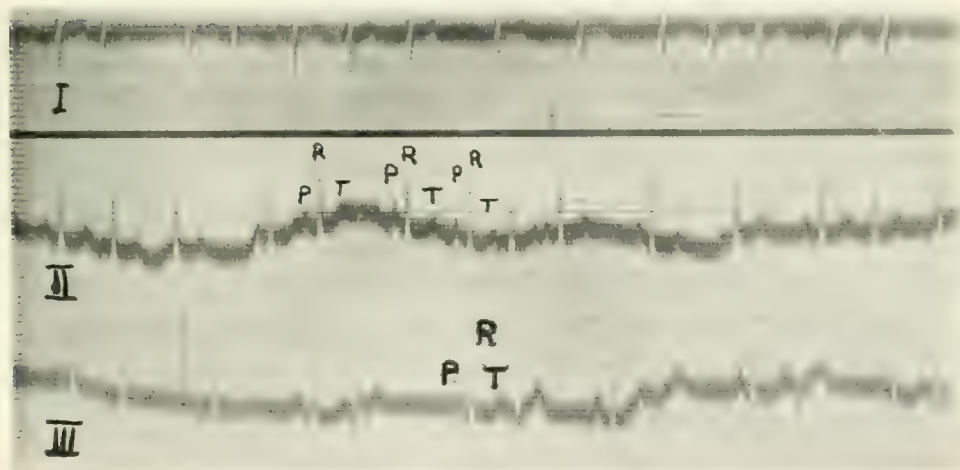


Fig. 2. Electrocardiogram taken July the 22nd, 1915, showing evidence of auricular activity. A well-marked *P* wave now precedes each ventricular complex. The rhythm is still irregular, the rate 96. In lead *III* four ventricular premature beats are seen of similar type to the one noted in Fig. 1. *R* and *T* waves similar to those in Fig. 1. Normal *P*-*R* interval.

OBSERVATIONS ON THE VITAL CAPACITY OF THE LUNGS IN CASES OF "IRRITABLE HEART."*

BY S. A. LEVINE AND F. N. WILSON.

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BREATHLESSNESS on exertion is perhaps the most constant and incapacitating symptom of which patients with "D.A.H." complain. In this respect they resemble cases of organic heart disease and methods employed in the study of dyspnoea in the latter may be used with advantage in the former. Lewis, Barcroft,¹ and their co-workers have already carried out investigations which suggest that the tendency to breathlessness in these men is, at least in part, the result of a reduction of the concentration of the buffer salts of the blood. In view of the importance of the condition, however, it seemed worth while to study the subject along other lines. We have therefore carried out a series of observations on the vital capacity in "D.A.H."

"D.A.H." (disordered action of the heart) is the official name in the British Army of a symptom-complex variously referred to in medical literature as "irritable heart," "soldier's heart," and "effort syndrome." The condition is a very common one in armies of the present war. The chief symptoms are breathlessness on exertion, precordial pain, palpitation, giddiness, weakness and general nervousness. In spite of the emphasis placed upon the heart by the names and the symptoms above mentioned, these patients show no evidence of structural disease of that organ. Tachycardia, precordial hyperalgesia, cyanosis of the hands, tremor, and excessive sweating are the principal findings on clinical examination. Most of the symptoms and signs are exaggerated by exertion. The present management of these cases is based upon a system of graded exercises. After an average stay of six weeks in the hospital, expeditionary force cases are classified, mainly in accordance with their exercise tolerance, for discharge into four groups. Class 1 includes men considered fit for full military duty; Class 2, those able to do moderate duty or full duty after further hardening; Class 3, men who can do only light sedentary work, and Class P.U., those to be discharged from the Army as *permanently unfit* for further military service.

* Undertaken on behalf of the Royal Medical Research Committee.

We decided to study the vital capacity in "D.A.H." cases, because of the relation between reduced vital capacity and dyspnœa found by Peabody³ in heart disease. It will considerably help a clear understanding of our findings if we briefly review some of the facts which he has established. In a study of about 140 healthy subjects he determined the average normal vital capacity and the magnitude of the individual variations. Since the vital capacity is defined as the greatest quantity of air which can be expelled from the lungs after a maximal inspiration, it will obviously vary with the size of the chest. Following earlier investigators, Peabody used height as a fair index of the size of the chest and a satisfactory basis for grouping his subjects. He divided normal males into three classes: those of 6 ft. in height or over had an average vital capacity of 5100 c.c.; those between 5 ft. 8½ in. and 6 ft., an average vital capacity of 4800 c.c.; and those between 5 ft. 3½ in. and 5 ft. 8½ in., an average vital capacity of 4000 c.c.. Ninety per cent. of an average figure was taken as the lower limit of normal, only one of the subjects in the three series falling below this limit. In a further study of heart disease he found a striking parallel between the severity of breathlessness and the percentage reduction of the vital capacity, and individual cases varied in their vital capacity with changes in the clinical condition.

Peabody believes that reduced vital capacity is an important factor in producing dyspnœa; his views may be briefly summarised as follows: If a patient 5 ft. 7 in. in height has a vital capacity of 2000 c.c., *i.e.*, 50% of the normal, and he is forced by exertion to breathe more air, he cannot increase the depth of respiration as much as a normal individual, and hence cannot increase his total ventilation to the same extent. An additional disadvantage of shallow breathing is that the dead space is a much greater portion of the inspired air than if each breath were deeper. Consequently he becomes dyspnœic more readily.

In this study we have used a spirometer of the ordinary cylindrical type, of about six and one-half litres capacity and graduated to read within 50 c.c.. From three to five observations were made on each subject and the highest reading was taken as the true vital capacity. In many instances further readings were taken of the same individual on different days during his stay in the hospital. In all, 131 patients were studied in this way. In 35 of these and in 10 normal individuals, observations were made on the effect of exercise on the vital capacity. In a few instances from 25 to 40 observations were made upon the same individual in rapid succession to note the effect of fatigue of the respiratory muscles upon the vital capacity.

We have used the class of discharge as an approximate index of the clinical condition and of the degree of breathlessness, for the two are generally parallel. We have, therefore, tabulated our observations upon the vital capacity in four tables corresponding to the four classes of discharge.

Twenty-two cases in Class 1 were examined (Table I). They are arranged in order of height. The percentage of the normal given in column four

represents the ratio between the observed vital capacity and Peabody's normal standard for the same height. The average of this group is 97%. Only four of the number fall below 90, which, as has been mentioned above, is considered the lower limit of normality. Two of the four have 89% and the other two have 83% and 85%. Briefly, it may be stated that 20 of the 22 cases in Class 1 have vital capacities falling practically within normal limits.

The results of studying 58 cases discharged in Class 2 are given in Table II. The average vital capacity of the entire group is 92% of the normal. This is appreciably lower than the average for Class 1. It is interesting to note that the average for the 17 cases which fall between the heights of 5 ft. 8½ in. and 6 ft. is only 88%, while the average of the 40 cases between 5 ft. 3 in. and 5 ft. 8½ in. is 93%. A similar difference in the averages of these two groups was found in all classes. (See Table V.) This may have been occasioned by the height of most of our patients between 5 ft. 8½ in. and 6 ft. falling in the lower half of the group. Nevertheless, it indicates the desirability of having normal standard figures for each varying inch of height. It is not altogether improbable that the normal standard determined for this group by Peabody is somewhat high.

The averages for cases discharged in Classes 3 and P.U. (permanently unfit) are 90% and 87% respectively. Thus there is a steady decline in the average vital capacity in cases of "D.A.H." as the severity of the condition increases: from 97% in those discharged in Class 1 to 87% in those discharged permanently unfit. While there are only 4 men of 22 in Class 1 who fell below the normal limit, there are 18 out of 58 in Class 2, 13 out of 26 in Class 3, and 15 out of 25 in Class P.U. These facts are summarised in Table V.

Peabody's view that diminished vital capacity causes dyspnoea has been criticised on the ground that dyspnoeic individuals cannot hold the breath sufficiently long to make a complete expiration, and that consequently breathlessness may cause low readings in determining the vital capacity. Although he has answered this criticism by pointing out that dyspnoeic subjects are able to hold the breath for several seconds immediately after the readings are made, it seemed desirable to determine the direct effect of dyspnoea upon vital capacity. A series of observations was therefore made upon 10 normal individuals. The vital capacity of each subject was taken immediately before and immediately after running up and down stairs until there was conspicuous breathlessness. The results of this experiment are shown in Table VI. Although the subjects could not hold the breath for many seconds, the average fall in vital capacity was but 2%. These figures offer strong evidence that dyspnoea *per se* does not diminish the vital capacity.

Although Peabody has shown that diminished vital capacity is probably an important factor in causing dyspnoea in heart disease and other clinical conditions, we do not feel justified in explaining the tendency to breathlessness

in "D.A.H." patients on the same basis. The difference between the average vital capacity of mild and severe cases is not sufficiently great; moreover, within a group of severe cases the degree of breathlessness on exertion and the percentage reduction of the vital capacity are not definitely parallel.

We are unable fully to explain the causes of the low average vital capacity found in the severer grades of "D.A.H." Certain suggestions may, however, be offered. Measurements of the chest after the method described by Lundsgaard and Van Slyke² were made in a few cases. They indicate that the volume of the chest was not the cause of our low readings. Pain in the left chest on deep inspiration is a not infrequent occurrence in severe "D.A.H." and this undoubtedly diminished the depth of the inspiration and so caused low readings in some of our cases. Other patients complained of giddiness upon deep breathing, and it is possible that this also, as well as other less defined subjective sensations on deep breathing, played some part in reducing the average vital capacity of the lower classes. The presence of such subjective sensations, as well as general nervousness, probably explains the great variability in successive readings observed in some of our patients.

The influence of general fatigue and exhaustion upon the vital capacity, so far as we know, has not been previously investigated. The forced inspiration and expiration of vital capacity experiments demand considerable effort on the part of the respiratory muscles. Any factor which impairs the efficiency of these muscles will have a tendency to lower the readings. The opposite is probably also true and may explain the unusually high readings which have been taken in athletes.

Patients with severe "D.A.H." complain of general weakness, and they are much more easily exhausted than normal people. It seemed to us that this might be a factor in causing the low average vital capacity observed in these cases. With this in mind we made readings on 35 patients before and after exercise. The results are summarised in Table VII. Two series of observations were made, one series testing the effect of prolonged exercise of 15 to 30 minutes, and the other the effects of exhausting exercise of short duration. In the first series, readings were made before and after the daily drills which the patients perform as a part of their routine treatment. In the second series readings were made before and after running up and down stairs until exhausted. Patients discharged in Classes 1 and 2 showed an average fall of 8% in the vital capacity as a result of the 15- to 30-minute drills, while those discharged in Classes 3 and P.U. showed a corresponding fall of 18%. The average fall in the vital capacity in 10 P.U. cases as a result of running up and down stairs until exhausted was 19%, while 10 normal individuals, as has been mentioned above, showed an average fall of only 2%. This difference becomes even more striking when one considers that the amount of exertion necessary to produce exhaustion was much less in the former than in the latter.

These figures indicate that fatigue may play a part in the reduction of the vital capacity in severe "D.A.H." In order to differentiate general fatigue from fatigue of the respiratory muscles, we asked some of the patients to sit quietly in a chair and to make maximal respiratory efforts in rapid succession so that approximately 30 readings were obtained in ten minutes. In normal controls the readings varied within very narrow limits. The mild "D.A.H." cases reacted in the normal way. The severer cases showed large variations, a few rapidly fell 25 to 50% or more, but the reaction was not sufficiently constant to allow us to draw definite conclusions as to the influence of fatigue of the respiratory muscles on vital capacity.

TABLE I.

Vital capacity in cases of "D. A. H." (Class 1).

No. of case.	Height.	Vital capacity.	% normal.	No. of case.	Height.	Vital capacity.	% normal.
102	6' 0½"	4350	85	82	5' 5½"	3550	89
95	5' 10"	4500	97	126	5' 5½"	3600	90
65	5' 8½"	4250	89	150	5' 5½"	4150	104
99	5' 8"	3700	93	86	5' 5"	3700	93
63	5' 8"	3800	95	101	5' 5"	3750	94
34	5' 7¾"	4700	117	4	5' 4¾"	4000	100
36	5' 7"	5250	131	48	5' 4½"	4100	102
81	5' 7"	3600	90	6	5' 4¼"	3850	96
100	5' 7"	3300	83	77	5' 4¼"	3600	90
33	5' 6½"	3800	95	123	5' 4"	4000	100
143	5' 6¼"	4000	100	AVERAGE			97%
112	5' 6"	4000	100				

TABLE II.

Vital capacity in cases of "D. A. H." (Class 2).

No. of case.	Height.	Vital capacity.	% normal.	No. of case.	Height.	Vital capacity.	% normal.
87	6' 2½"	5400	106	146	5' 6½"	4400	110
94	5' 11"	4400	92	74	5' 6"	3000	75
130	5' 10½"	3800	79	96	5' 6"	3500	88
38	5' 10½"	4600	96	128	5' 6"	3500	88
114	5' 10"	4300	90	145	5' 6"	3850	96
109	5' 10"	3700	77	147	5' 6"	3700	93
24	5' 9¾"	4650	97	45	5' 5¾"	3850	96
137	5' 9¾"	4300	90	69	5' 5¾"	3600	90
62	5' 9¾"	4300	90	79	5' 5¾"	3850	96
17	5' 9¾"	3600	75	88	5' 5¾"	3600	90
62	5' 9¾"	4300	90	75	5' 5¾"	3700	93
64	5' 9"	3750	78	3	5' 5½"	3350	84
11	5' 9"	3950	82	8	5' 5½"	3500	88
106	5' 9"	4100	85	19	5' 5½"	3600	90
115	5' 9"	5000	104	30	5' 5½"	3650	91
40	5' 8½"	4500	94	35	5' 5½"	3800	95
84	5' 8½"	4450	93	70	5' 5½"	3650	91
18	5' 8½"	4150	87	68	5' 5"	3550	89
127	5' 8¼"	4000	100	47	5' 5"	3350	84
76	5' 8"	4050	101	1	5' 4½"	3800	95
120	5' 8"	4700	116	20	5' 4½"	3650	91
116	5' 7½"	3400	85	31	5' 4½"	3600	90
83	5' 7¼"	3800	95	78	5' 4¼"	2900	73
104	5' 7"	3600	90	122	5' 4"	4000	100
105	5' 7"	3500	88	60	5' 4"	3950	99
118	5' 7"	3800	95	29	5' 3¾"	4000	100
158	5' 7"	3700	93	41	5' 3¾"	3100	78
7	5' 7"	3400	85	117	5' 3"	4000	100
21	5' 6½"	4150	104	AVERAGE 92% ₀			
144	5' 6½"	3950	99				

TABLE III.
Vital capacity in cases of "D. A. H." (Class 3).

No. of case.	Height.	Vital capacity.	% normal.	No. of case.	Height.	Vital capacity.	% normal.
110	6' 0"	5350	105	138	5' 6"	2800	70
178	5' 11"	3200	67	129	5' 5 $\frac{1}{4}$ "	3500	88
80	5' 10 $\frac{1}{2}$ "	4600	96	2	5' 5 $\frac{1}{2}$ "	3000	75
113	5' 10"	3800	79	67	5' 5 $\frac{1}{4}$ "	3900	98
12	5' 9 $\frac{3}{4}$ "	3800	79	149	5' 5"	3000	75
103	5' 9"	3700	77	16	5' 4 $\frac{3}{4}$ "	4050	101
10	5' 9"	4050	84	97	5' 4 $\frac{3}{4}$ "	3700	93
23	5' 8 $\frac{1}{4}$ "	4600	115	37	5' 4 $\frac{1}{2}$ "	3300	83
49	5' 8"	3300	83	43	5' 4 $\frac{1}{4}$ "	3500	88
27	5' 7 $\frac{3}{4}$ "	4200	105	53	5' 4 $\frac{1}{4}$ "	3600	90
22	5' 7 $\frac{1}{4}$ "	4300	108	135	5' 4"	3900	98
139	5' 7"	4300	108	125	5' 3 $\frac{1}{4}$ "	3300	83
46	5' 6 $\frac{1}{4}$ "	3650	91				
107	5' 6"	3700	93				
				AVERAGE 90%			

TABLE IV.
Vital capacity in cases of "D. A. H." (P. U. Class).

No. of case.	Height.	Vital capacity.	% normal.	No. of case.	Height.	Vital capacity.	% normal.
16	5' 11½"	3800	79	91	5' 7"	3000	75
132	5' 11"	3900	81	121	5' 7"	3900	98
159	5' 10½"	3100	65	18	5' 7"	4000	100
170	5' 9½"	4150	86	92	5' 6½"	2800	70
168	5' 9"	4150	86	98	5' 6"	3000	75
151	5' 8½"	3900	81	72	5' 5¾"	3550	89
156	5' 8½"	4000	83	111	5' 4½"	3500	87
12	5' 8"	4300	108	136	5' 4½"	3800	95
14	5' 8"	3300	83	108	5' 3½"	3600	90
161	5' 7½"	4000	100	10	5' 3½"	3000	95
93	5' 7½"	3800	95	6	5' 3"	3700	93
8	5' 7½"	3650	91	20	5' 3"	3350	84
162	5' 7¼"	3800	95	AVERAGE 87.0			

TABLE V.

Summary of Tables I to IV.

Class.	ALL CASES.				Cases of 5' 8½'' to 6' high.		Cases of 5' 3'' to 5' 8½'' high.	
	No. of cases.	Average vital capacity.	Number below 90% normal.	% below 90% normal.	No. of cases.	Average vital capacity.	No. of cases.	Average vital capacity.
1	22	97%	4	18	2	93%	19	98%
2	58	92%	18	31	17	88%	40	93%
3	26	90%	13	50	6	80%	19	92%
P.U.	25	87%	15	60	7	80%	19	89%

TABLE VI.

Normal exercise response.

No. of case.	Height.	Vital capacity before.	% normal.	Vital capacity after.	% normal.	Pulse before.	Pulse after.
163(b)	5' 11¾''	4075	85	3900	81	114	168
165(d)	5' 10½''	5000	104	4800	100	100	156
164(l)	5' 10¼''	4000	83	3750	78	84	152
165(c)	5' 9''	5200	108	5000	104	60	148
165(b)	5' 8¼''	3550	89	3600	90	60	136
165(a)	5' 8''	3850	96	3700	93	87	156
176	5' 7''	3750	94	3650	91	78	174
163(a)	5' 6''	4000	100	4000	100	68	132
165(e)	5' 6''	3800	95	3700	93	68	156
164(a)	5' 4½''	3900	98	3800	95	84	128
	Average	..	95	..	93	80	151

TABLE VII.

Effect of exercise on vital capacity in cases of "D. A. H."

DRILLS.

Class.	No. of cases.	Average vital capacity before.	Average vital capacity after.	Fall due to drill.
1 and 2	17	89%	81%	8%
3 and P.U.	8	75%	57%	18%

EXHAUSTING EXERCISE.

Class.	No. of cases.	Average vital capacity before.	Average vital capacity after.	Fall
P.U.	10	83%	64%	19%

SUMMARY.

The average vital capacity is slightly but definitely reduced in the severer cases of "D.A.H." The reduction is not sufficiently constant to be of use in classifying these patients, nor is it sufficiently great to account for the breathlessness which they show so readily on exertion. The slight reduction in the vital capacity is due partly, if not altogether, to the discomfort which the severe cases experience on deep breathing and to fatigue.

Exercise considerably reduces the vital capacity in severe "D.A.H." But since exercise, sufficiently strenuous to produce breathlessness in normal individuals, does not materially reduce vital capacity in these, breathlessness cannot be regarded as the cause of reduced vital capacity in severe "D.A.H." cases.

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THE BICARBONATE CONCENTRATION OF THE BLOOD PLASMA IN CASES OF "IRRITABLE HEART."*

By FRANK N. WILSON, SAMUEL A. LEVINE AND A. B. EDGAR.

(From the Military Heart Hospital, Colchester.)

FROM a study of the vital capacity of the lungs in cases of "irritable heart," the details of which are published in an accompanying paper, we concluded that the breathlessness on exertion which these patients experience is not caused by a reduction of the vital capacity. It was desirable, therefore, to determine whether other common causes of breathlessness were present. It is now recognised that one of the important causes of breathlessness is "acidosis," a condition in which, either because of the increased production or the decreased elimination of acid substances, there is a decrease in the alkaline reserve of the blood. We decided, therefore, to determine whether this condition is present in cases of "irritable heart" who present this symptom. Of the many methods which have been used in estimating "acidosis" we chose the Van Slyke-Cullen method¹ for the determination of the bicarbonate concentration of the plasma as the most suitable and convenient for our purpose. It demands no co-operation on the part of the patient, an important consideration in dealing with "irritable heart" cases, and it can be carried out with few laboratory facilities.

It may be well to give a short outline of the method here. Blood is drawn from the median vein of the arm without stasis, oxalated to prevent clotting, immediately centrifugated, and the plasma separated from the cells. About 3 c.c. of plasma is then saturated in a large separating funnel with alveolar air (5.5% carbon dioxide). One c.c. is introduced into the Van Slyke-Cullen apparatus, acidified, and the gas extracted by means of a Torricellian vacuum and measured over mercury. From the amount of gas extracted under known conditions of temperature and pressure the amount of carbon dioxide (reduced to zero degrees and 760 mm. Hg. pressure) bound as bicarbonate by 100 c.c. of plasma at 20 degrees can be determined by a formula or by a table prepared by the originators of the method. According to Van Slyke and Cullen the value determined by this method indicates not only the alkaline reserve of the blood but that of the whole body, and the method is stated to be a very sensitive one for the detection of "acidosis."

* Work undertaken on behalf of the Royal Medical Research Committee.

We have examined 19 cases of "irritable heart" by this method. Fourteen of these were severe cases which were to be discharged from the Army as permanently unfit for further military service or which were to be sent out in the lowest duty category. With the exception of *Case 4*, all of these patients were conspicuously short of breath on slight exertion. The remaining five patients were taken as controls. For this purpose we selected men who were not short of breath and who were to be discharged as fit for full military duty. The results of this study are tabulated below. A short summary of the main clinical features of each case, the method used in drawing the blood and the amount of carbon dioxide bound as bicarbonate by 100 c.c. of plasma are given.

TABLE I.

Case 1.	Conspicuous dyspnoea on light exercises. Symptoms came on after pyrexia of unknown origin. Discharged as fit only for sedentary employment.	Blood drawn with syringe and delivered into centrifuge tube.	Amount of CO ₂ . 68.8
Case 2.	Had been short of breath as long as he could remember; was able to do 30-minute exercises. Discharged as fit only for sedentary employment.	" "	66.9
Case 3.	Complained of dyspnoea, precordial pain, and weakness, and dated these symptoms from childhood. Showed conspicuous dyspnoea on 15-minute exercises. Discharged as permanently unfit for service.	" "	71.3
Case 4.	Was not able to do 15-minute exercises and was discharged as permanently unfit. Symptoms came on after an infection of the hand. No conspicuous dyspnoea.	" "	67.3
Case 5.	Chief complaint was shortness of breath which came on after "burial" by a shell. Discharged from the Army as permanently unfit for service.	" "	62.5
Case 6.	Was conspicuously breathless on slight exertion. Symptoms developed gradually while on active service. Discharged as permanently unfit for service.	" "	68.1
Case 7.	Very conspicuous dyspnoea after 15-minute exercises. Dated symptoms from rheumatic fever. Discharged as permanently unfit for service.	Blood drawn with syringe and delivered under oil.	69.5
Case 8.	Conspicuous dyspnoea on light 15-minute exercises. Dated symptoms from trench fever. Was invalided for rheumatic fever. Discharged as permanently unfit for service.		74.8
Case 9.	Conspicuous dyspnoea on light 15-minute exercises. Symptoms developed during training. Discharged as fit only for sedentary employment.	" "	75.0
Case 10.	Conspicuous dyspnoea on light 15-minute exercises. Symptoms dated from gassing. Discharged as permanently unfit for service.	" "	75.0
		" "	66.8

TABLE I—*continued*.

Case	Description	Blood drawn directly into centrifuge tube.		Amount of CO ₂ .
		No oil used.		
Case 11.	Very conspicuous dyspnoea at rest which was absent during sleep, and was probably a neurotic dyspnoea. Could not do light exercises and was transferred to a hospital for functional nervous diseases.			
Case 12.	Conspicuous dyspnoea on light exercises. Symptoms came on during training. Discharged as permanently unfit for further service.	69.2
Case 13.	Conspicuous dyspnoea on slight exertion. Symptoms came on after influenza. Is still in hospital and is still improving.	70.3
Case 14.	Conspicuous dyspnoea on light exercises. Was weak as a boy, but dated symptoms from malaria. Discharged as permanently unfit.	70.6
CONTROLS.				
Case 15.	All these patients showed a practically normal exercise tolerance. They were not short of breath, and were discharged as fit for full duty.	Blood drawn directly into centrifuge tube.		69.2
		No oil used.		
Case 16.		71.1
Case 17.		72.0
Case 18.		70.0
Case 19.		71.0

According to Van Slyke and Cullen the normal lower and upper limits of plasma carbon dioxide capacity as determined by this method are 53 and 78 c.c. per 100 c.c., respectively. Our results lie between 62.5 and 75 c.c., and are, therefore, within normal limits. In two cases, 8 and 9, the results, 75 c.c. in each case, are near the upper limit of normality. This was probably partly due to the manner in which the blood was drawn, for, when the blood is delivered under oil so that no carbon dioxide is allowed to escape, the results are somewhat higher than when the blood is momentarily exposed to the air at the time of its withdrawal from the vein. This fact is well illustrated by *Case 7*, in which both methods were used; the sample delivered under oil showed a carbon dioxide capacity 5.3 volume per cent. greater than the other where no oil was used. There was no difference in the carbon dioxide capacity of the blood between severe cases which showed conspicuous dyspnoea and control cases which showed none. We believe that we are justified in concluding from this series of cases that there is no definite reduction of the alkaline reserve of the blood in cases of "irritable heart" and that the tendency to breathlessness in this condition is not due to the presence of "acidosis."

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THE PISTOL-SHOT SOUND IN AORTIC DISEASE.*

BY ROSS A. JAMIESON AND FRANK N. WILSON.

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IN a recent article entitled "A study of arterial sounds,"¹ Stewart R. Roberts states that the femoral sounds heard in aortic insufficiency are due to a transmission of the heart sounds through the blood column, and that the sound over the brachial artery heard in taking the blood pressure is due to transmission from closure of the aortic valve. Neither statement is supported by accurate observations upon these sounds, for no graphic records were made. It seems highly improbable that either of these sounds could be transmitted from the heart, and a careful study of the femoral sounds quickly proves that they are associated with the pulse wave in the artery and have nothing whatever to do with the heart sounds.

Although systolic sounds in the femoral artery are not confined to aortic insufficiency, yet they are very frequent and very loud in this condition and the present work was therefore undertaken on patients with this heart lesion. Individuals were chosen in whom the pistol-shot sound was not confined to the femoral, but was also heard in the dorsalis pedis or in the posterior tibial artery. In each case four sets of simultaneous records were taken: first, the electrocardiogram (lead *II*) and simultaneously the pistol-shot sound in the femoral; second, the electrocardiogram and the pistol-shot sound in the dorsalis pedis or in the posterior tibial; thirdly, the electrocardiogram and the pulse wave in the femoral; and fourthly, the electrocardiogram and the pulse wave in the dorsalis pedis or posterior tibial, whichever had been previously chosen.

In taking the simultaneous records of the electrocardiogram and the pistol-shot sounds, a Cambridge string galvanometer with a double string carrier was used, the sounds being changed into electrical vibrations by means of a microphone in the usual way. In recording the pulse wave a Mackenzie polygraph was used, the writing lever being so arranged that it moved in front of the slit of the galvanometer camera so that the record was taken photographically instead of upon polygraph paper. The transmission time of the rubber tubing and capsules used in recording the pulse wave was determined and was allowed for on each occasion in making measurements.

*Undertaken on behalf of the Royal Medical Research Committee.

With these four sets of simultaneous records at hand it was possible to determine accurately the time elapsing between the apex of the *R* wave of the electrocardiogram, which was used as a standard, and the appearance of the pistol-shot sound or the pulse wave at the femoral artery (or the tibial or dorsalis pedis artery, as the case might be). The distance from the point used over the femoral artery to the point used over the dorsalis pedis was also measured with the idea of comparing it with the difference in time between the appearance of the pistol-shot sound at these two points. Now if, as Roberts states, the pistol-shot sound is transmitted from the heart, we should expect it to be heard at the femoral and at the dorsalis pedis at almost the same instant, for the distance between these two points is only about eighty centimeters and, although we do not know exactly the speed with which sound is transmitted along the blood column in the arteries, still it must be of the same order of magnitude as the speed of transmission in air which is about 1,800 feet per second. Furthermore, we should not expect to find any constant or definite relationship between the appearance of the pistol-shot sound and the appearance of the pulse wave at the femoral and at the dorsalis pedis, for clearly sound transmission is very many times more rapid than is pulse transmission.

Actual observation showed that the pulse wave and the pistol-shot sound were always closely associated, the sound coming very nearly with the onset of the first up-stroke of the pulse. We also found that a considerable interval of time elapsed between the appearance of the sound at the femoral and its appearance at the dorsalis pedis; an interval so great that, if conducted as sound, its speed of conduction would have been about 1,000 cm. per second, an impossibly low speed for sound conduction, the known speed for pulse conduction. This finding, together with the association of the sound and the pulse wave spoken of above, allow of only one conclusion: the pistol-shot sound is not transmitted from the heart but is produced, at the point where it is heard, by the impact of the pulse wave. Indeed it seems strange that anyone could have imagined otherwise.

An example of the curves obtained is shown in Fig. 1. The separate curves are so arranged in the figure that simultaneous points fall in the same vertical line. The polygraphic curves are shifted to the left by 1/30 second in order to allow for the time of transmission through the rubber tubing used. The measurements of this and of four other sets of curves are given in Table I in decimal fractions of a second. The five columns in this table are as follows:—the first column gives the time from *R* of the electrocardiogram to the occurrence of the sound in the femoral artery, the second column gives the time from *R* to the appearance of the pulse wave in the femoral, the third and fourth columns give the times from *R* to the occurrence in the dorsalis pedis (or posterior tibial) of the sound and the pulse wave, respectively, and the fifth column the hypothetical speed of transmission of the sound from the femoral to the dorsalis pedis as determined by comparing the distance with the time interval as previously described.

TABLE I.

No.	<i>Time from "R" to occurrence of</i>				Hypothetical speed of transmission of sound from femoral to dorsalis pedis.
	Sound at femoral.	Pulse wave at femoral.	Sound at dorsalis pedis.	Pulse wave at dorsalis pedis.	
1	·192 sec.	·180 sec.	·325 sec.	·326 sec.	1151 cm. per sec. 952·4 cm. "
2	·178 "	·179 "	·244 "	·256 "	
3	·167 "	·215 "	·251 "	·281 "	
4	·171 "	·165 "	·258 "	·253 "	
5	·169 "	·156 "	·277 "	·263 "	

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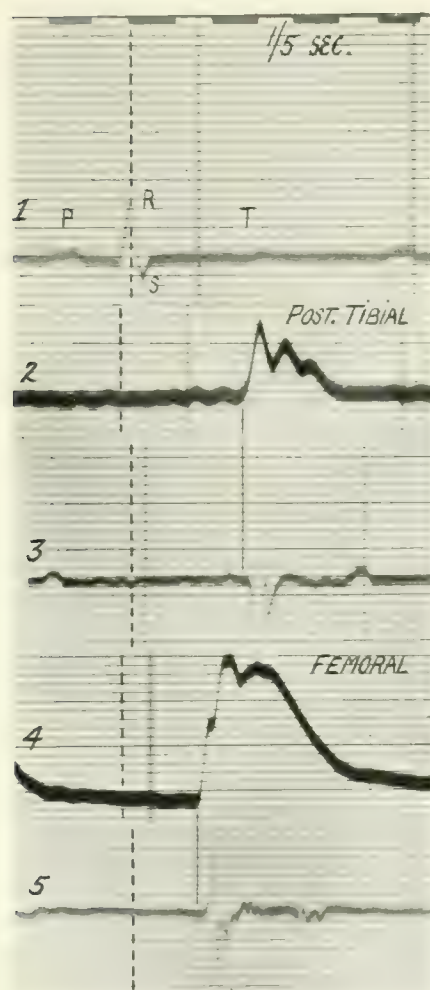


Fig. 1. Graphic records of Case 5.

1. Electrocardiogram lead II.
2. Pulse wave in posterior tibial artery.
3. Pistol-shot sound in the posterior tibial artery.
4. Pulse wave in femoral artery.
5. Pistol-shot sound in femoral artery.

The records are so placed that simultaneous points fall in the same vertical line. The polygraphic records are shifted to the left by a thirtieth of a second, to allow for the transmission time of the tubing and capsule used in recording them.

MUSICAL DIASTOLIC MURMURS IN AORTIC INSUFFICIENCY.*

By FRANK N. WILSON AND ROSS A. JAMIESON.

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EMPHATIC musical murmurs are comparatively rare. Systolic murmurs maximal at the apex occasionally have a faint musical quality, but loud and clear cut musical murmurs are almost always best heard at the base, usually in the aortic area. Such murmurs are usually diastolic in time but musical systolic murmurs are occasionally heard. These musical murmurs are thought to be produced in one of three ways, by the rupture of a valve segment, by the perforation of a valve segment, or by a cord-like strand of tissue across the valve orifice. They are most frequently discovered in cases where there is a suspicion of valve rupture. There is often the history of some accident which may have brought this about, and it is thought that the rupture occurs as the result of the violent muscular effort made in attempting to recover the balance after a fall or a blow. Experiments upon the pressure necessary to rupture healthy valve segments make it seem improbable that healthy valves ever rupture as the result of any accident, but when rupture occurs it is rather to be assumed that the valve segment was already weak because of organic disease such as syphilis or sclerotic changes.

Perforation of a valve may occur in the same circumstances as rupture or as the result of an ulcerative process, or it may be a congenital defect; in some cases an aneurism of the valve first forms and subsequently ruptures.

During the last six months at the Sobraon Military Hospital, three cases showing the signs of aortic incompetence with loud musical diastolic murmurs have been discovered. Two of these patients gave a history of being "blown up" by the explosion of a shell. The murmur became audible to each a considerable time after the occurrence of the accident, five weeks in *Case 1*, four months in *Case 3*. It is, therefore, impossible to say definitely whether the accidents were in any way responsible for the valve lesions. In neither case was there any history or evidence of organic disease which might have weakened the valve. The third case (*Case 2*) became conscious of the murmur on the fourth day after he was gassed with phosgene, but there was no history of any physical injury or concussion,

* Work undertaken on behalf of the Royal Medical Research Committee.

and since he gave a history of chancre and his complement fixation test was positive, his valve lesion may be attributed to syphilis. The murmurs were recorded graphically by means of the phono-cardiograph. They were all sufficiently loud to be audible to the patients themselves and to the unaided ear of the examiner at a distance of from one to five feet from the chest wall. They were decrescendo in type. In *Case 1* the musical murmur filled the entire diastole, but in the other two cases only the first part of the murmur was musical, the latter part being heard as a high-pitched blow. The murmurs were very variable in intensity, especially in *Cases 2* and *3*, and they were exaggerated by exertion. In *Case 3* the murmur was exaggerated by conversation also. The graphic records are shown in Figs. 1, 2 and 3. The vibration frequency in these records ranges from about 115 to 175 per second, but judging from the musical pitch of the murmur, overtones of a much higher frequency were probably present. The detailed histories follow :—

Case 1. Pte. N., a butcher, aged 45, was admitted to the Sobraon Military Hospital on the 26th day of February, 1918, with the diagnosis of valvular heart disease. He was strong and healthy as a boy and played football and cricket without difficulty. He joined the Army in 1901 at the age of 28, and served in South Africa and in India until 1912. During this time he was well, with the exception of malaria and enteric fever in 1904. He gave no history of rheumatic fever, chorea or tonsillitis, and denied venereal disease. In November, 1914, he re-enlisted and was sent to France in the infantry, where he did full duty without trouble until May, 1916. At this time he was buried and wounded by the explosion of a shell, and was sent to the hospital unconscious. He did not know how long he was unconscious, and could not remember anything that happened during the first five weeks in hospital. At the end of that time he became aware of a musical “whizzing” sound in his chest and of pain in his side and head. After five or six months in hospital he was discharged by his medical officer as fit for light duty. He carried on in a labour company with some difficulty until January, 1918, when he was examined by a medical board and recommended for hospital treatment. He complained of shortness of breath, dizziness and precordial pain, and dated these symptoms from his “burial.”

Physical examination was negative except for the heart. The apex beat was located in the 5th interspace $11\frac{1}{2}$ cm. from the mid-line. A diastolic thrill was felt over the entire precordium; it was maximal in the aortic area. On auscultation a very loud musical diastolic murmur was heard. It was loudest in the aortic area, but was widely transmitted over the chest. It was decrescendo in type and filled the entire diastole. In a quiet room the murmur could be heard with the unaided ear at a distance of four feet from the chest. It was somewhat louder after exercise. No pistol-shot sound or other vascular signs of aortic insufficiency were elicited. The complement fixation test was negative.

The phono-cardiographic records show a diastolic murmur beginning immediately after the second sound and ending with the first sound. The amplitude of the vibrations varies considerably, but there is a gradual diminution in amplitude from the beginning to the end of the murmur, and this change in amplitude is most rapid in mid-diastole, so that the amplitude of the first half of the murmur is about twice as great as that of the last half. There is also a decrease in the vibration frequency of the murmur with this change of intensity; the frequency for the first half is 150 and for the last half only 125. The murmur is fairly constant in intensity from cycle to cycle.

Case 2. Pte. G., a brewery cellarman in civil life, aged 42 years, was admitted to the Sobraon Military Hospital on March the 30th, 1918, with a diagnosis of valvular heart disease and gas poisoning. He said that as a boy and young man he had been quite fit and had played strenuous games, football, etc., without difficulty. When seven years of age he had pleurisy. He had never suffered from any form of rheumatism or other infection. He admitted to having had a "soft chancre" in June, 1902, for which he received treatment in hospital for fifteen days, and he apparently never developed any later symptoms.

He enlisted in the regular Army in 1895, and served for eight years in England, South Africa and India. While in South Africa he had a mild attack of dysentery, and in India contracted malaria, but was never in hospital for either condition. In 1902 he was discharged, time-expired. After discharge he went to work as cellarman in a brewery and continued this work for the following 12 years. The work was heavy, and it was at this time that he first noticed some shortness of breath, palpitation and dizziness on heavy exertion, but he was always fit for his work. The above-mentioned symptoms persisted, but apparently did not increase in severity.

In April, 1916, he enlisted in the infantry. He had three months training in England, experiencing some difficulty and frequently falling out on account of shortness of breath, this being his only complaint. In August, 1916, he was sent to France fit for general service, and a few weeks later went up the line on full duty. Three weeks after this he complained of a feeling of tightness in his chest, shortness of breath and cough. He was given lighter duty as a cook and carried on with his work until March, 1918. During this period he said his condition was as nearly as he could tell about as it had been in civil life, with the one exception that for the last five months of this period he had been more conscious of his heart beat. In March, 1918, he was gassed (phosgene) and three days later he reported sick with loss of voice, tightness across chest, shortness of breath and cough. He was then sent into hospital and on the following day he was conscious for the first time of a noise in his chest which had since persisted and which he described as "like water rushing through a pipe." The patient was conscious of this noise only when he was in the horizontal position. There was no history of any injury or concussion.

The physical examination showed the patient to be a man of fairly good physique. He was pale and slightly cyanosed. The respiratory, digestive, nervous and genito-urinary systems revealed nothing abnormal on routine examination. The apex beat was in the 4th interspace, the outermost point of pulsation being 11 cm. from the mid-sternal line and just in the nipple line. On percussion there was evidence of moderate enlargement of the heart extending both to the right and left.

At the apex the first sound was booming in quality and followed by a harsh murmur. The second sound at this area was blurred and followed by a distant blowing murmur. At the base both to the right and left of the mid-sternal line the second sound was accentuated. In this locality a short rather harsh systolic murmur was audible and was transmitted in an upward direction. At the base of the heart and of maximum intensity over the third left interspace a diastolic murmur was heard. This murmur followed immediately on the second sound and in its first part was high-pitched and of musical quality. This musical quality was retained during approximately the first half of diastole and then gradually faded away into a blowing diastolic murmur. This musical murmur was loudest when the patient was lying down, though it was also audible when he was in erect position. It was transmitted to the entire chest posteriorly but could be best heard over the left upper portion of the chest. Both the systolic and diastolic murmurs were transmitted to the carotid arteries but were of greatest intensity in the right carotid. Respiration had no appreciable effect on the character of the musical murmur.

The pulse was of the water-hammer variety, capillary pulsation was present, and the pistol-shot sound could be heard over the femoral and dorsalis pedis arteries. The systolic blood pressure was 135 mm. Hg., and the diastolic 45, fading off to 0. The complement fixation test was positive.

The phono-cardiographic records show a murmur usually occupying the entire diastolic period, but occasionally stopping just short of the first sound. The murmur is decrescendo in type, with a very rapid decline in amplitude at the end of the first third of diastole. The vibration frequency for the first half of the murmur is considerably greater (140) than for the second half (115). There was considerable variation in the murmur from cycle to cycle. At times the murmur almost disappeared and at other times the greater frequency and amplitude of the first part of the murmur persisted almost to the end of diastole.

Case 3. Pte. S., a dock labourer in civil life, aged 34 years, was admitted to the Sobraon Military Hospital on March the 8th, 1918, with a diagnosis of valvular heart disease. He gave a history of having been fit and robust as a child. When eight years of age he left school and went to work on a farm and at the age of 11 was doing heavy farm work. At the age of 17 he commenced work as a dock labourer. This entailed for the most part very heavy physical exertion, and he continued at this occupation until joining the Army. In addition to his work he played

football until he was 26 years of age and then discontinued the game on account of lack of time and disinclination.

He had whooping cough when a child : he had never suffered from any form of rheumatism, and he denied having had venereal disease of any kind.

He enlisted on December the 31st, 1914, in the Army Service Corps, and one week later he was sent to France for general service and put to work on the railway, unloading supplies, munitions, etc.. The work was heavy, but he carried on without any difficulty whatever. In April, 1916, he was slightly gassed. He continued work, although he did not feel very fit. After two weeks, however, he said he felt just as well as ever.

Early in February, 1917, he had an injury to the great toe of the left foot which later necessitated incision and removal of the toe-nail under general anaesthesia. He returned to full duty in April and felt quite fit. Early in June, 1917, a large shell exploded within a few feet of him : the concussion threw him off his feet, but in so far as he knew he was not physically injured. After this he was very nervous and had frequent attacks of palpitation. Because of these symptoms he was moved back to a position about fifteen miles behind the line, but was kept on heavy work. He now found the work difficult because of occasional palpitation and dizziness. He continued doing heavy work, however, until the end of November, 1917. At this time he first became conscious of a noise in his chest. This noise might continue for five minutes and then disappear for an hour or more. He observed that any especially strenuous work increased it. The noise in the chest, which the patient described as being at first of a wheezing character, slowly increased in intensity and frequency, reaching its maximum one week after its first appearance. At this time also he first noticed that he was short of breath on exertion. He continued work for several weeks : then on December the 24th went sick, complaining chiefly of breathlessness. He was sent into hospital, but after being detained for three days was returned to light duty. For the following two months he did practically no work and he was returned to hospital on February the 26th.

Physical examination. The patient was well developed ; there was slight pallor and slight cyanosis. The respiratory, digestive, nervous and genito-urinary systems revealed nothing abnormal on routine examination. The apex beat was in the 5th left interspace, 11 cm. from the mid-sternal line and just outside the nipple line. There was a slight degree of cardiac hypertrophy. A conspicuous systolic pulsation was seen in the vessels of the neck. On palpation over the precordium a diastolic thrill of inconstant intensity was felt along the left border of the sternum. On auscultation at the apex a short rather harsh systolic murmur was heard. The second sound at this area was accentuated and was followed by a blowing, rather distant murmur. This murmur was audible at all areas, but of maximum intensity over the third left interspace close to the sternal margin. It was frequently associated with a high-pitched, humming musical murmur of very

variable character and of varying intensity. When present it followed immediately on the second sound and was of maximum intensity at its inception. Frequently this murmur would fill the entire diastolic period, commencing with a booming sound and fading off to a hum. In other cycles the musical murmur would occur immediately after the second sound and then quickly fade off into the blowing diastolic murmur. In yet other cycles only the blowing diastolic murmur was heard. This musical murmur was audible with the patient in either the erect or horizontal position, but was louder when he was lying. It was increased by exertion, by excitement, and even by conversation. It could be readily heard in a quiet room at a distance of 8 inches from the chest. The musical murmur could be heard over the entire chest anteriorly and posteriorly.

On full inspiration the musical character of the murmur disappeared although the diastolic blow persisted. On full expiration the musical character was quite markedly intensified.

The second sound was accentuated over the base of the heart. In the region of the second right costal cartilage a short distant systolic murmur was heard. The radial pulses were equal and of the water-hammer variety and capillary pulsation was present. No definite pistol-shot sound was made out over the arteries.

The systolic blood pressure was 140 mm. Hg., and the diastolic 60 mm. Hg., fading off to 0. The sound of the musical murmur could be heard very distinctly below the pressure cuff in the left arm and was not obliterated by a pressure of 250 mm. Hg.. This sound was much less distinctly heard in the right arm than in the left.

The complement fixation test was negative.

The graphic records show a diastolic murmur beginning immediately after the second sound. It begins with an abrupt crescendo followed by a gradual but irregular decrescendo. The vibration frequency for the first half of the murmur is 170, while that of the last half is 130. There was a great variability in the intensity of the murmur from cycle to cycle. In some of the cycles no vibrations could be seen, while in others only a few vibrations immediately following the second sound were recorded.

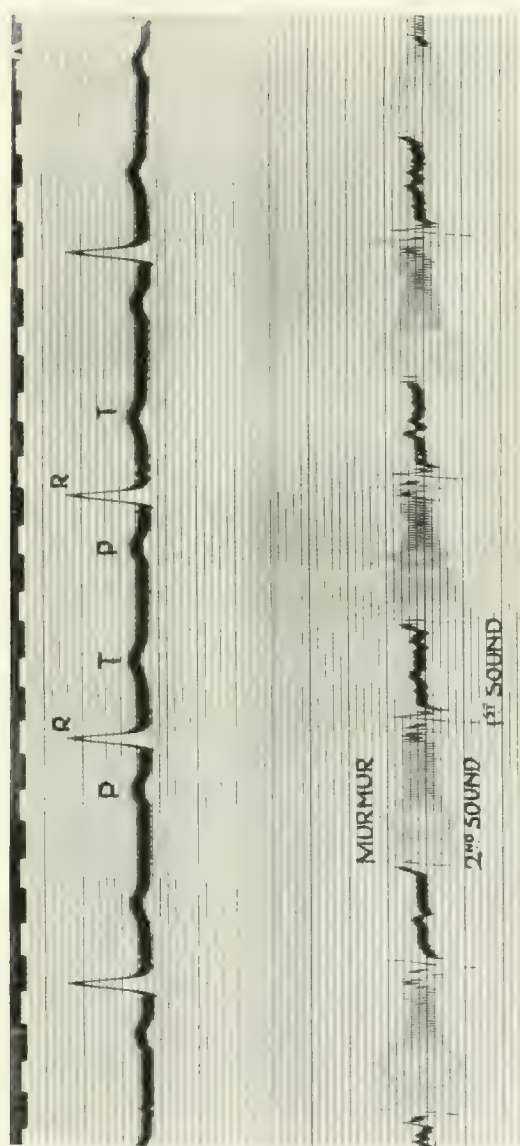


Fig. 1. (Case 1.) A decrescendo diastolic murmur beginning immediately after the second sound and ending with the first sound. The vibrations of the first half of the murmur are of greater amplitude than those of the last half. The vibration frequency of the first half of the murmur is 150 per second; and of the last half 125 per second. Time marker in fifths of a second.

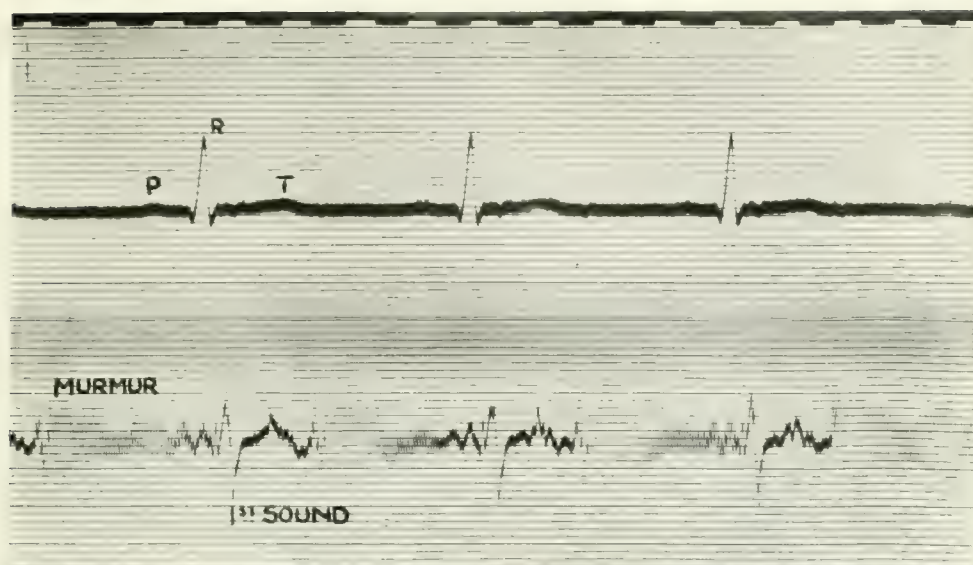


Fig. 2. (Case 2.) A decrescendo diastolic murmur showing considerable variation in intensity and duration from cycle to cycle. The vibration frequency of the first half of the murmur is 140 and of the last half 115.

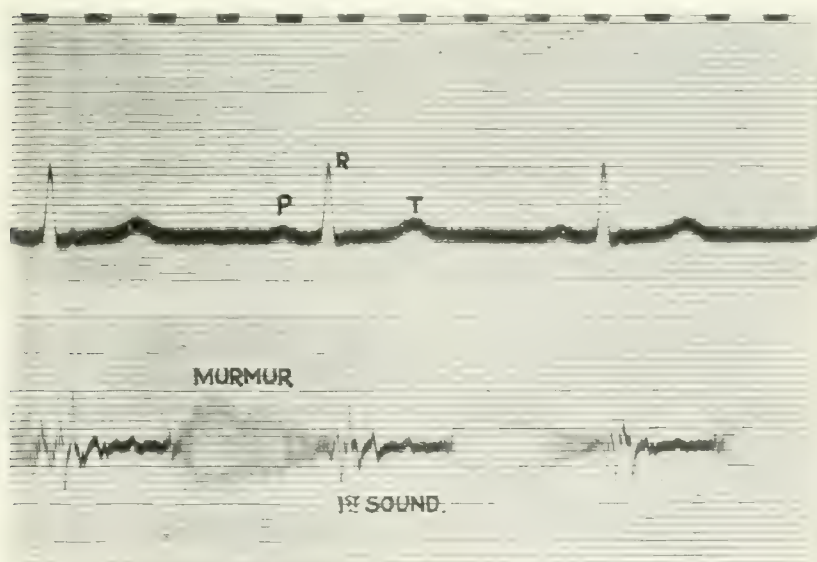


Fig. 3. (Case 3.) A diastolic murmur beginning with an abrupt crescendo followed by a gradual decrescendo. The vibration frequency of the first half of the murmur is 170, and of the last half 140. Other records show great variations in the intensity of the murmur and in its duration: the murmur is entirely absent from some cycles and only occupies the first part of diastole in others.

TIME RELATIONS OF HEART BEATS.

*Respiratory Variations of Heart Rate in the Presence of Auricular Fibrillation.**

By EUGENE S. KILGORE.

(*University of California.*)

THAT in the extensive literature of "absolute arrhythmia" no report has been made heretofore of the presence or absence of coincident respiratory variations in the heart rate is due undoubtedly to the over-shadowing influence of other factors in these cases. The pulse waves of these patients certainly give the impression of a quite random distribution, and it is impossible even on close examination of tracings to detect a constant relation between phases of respiration and the longer or shorter pulse intervals. It is conceivable, however, that periodic retarding or accelerating influences, if comparatively slight, may be concealed behind an otherwise absolute arrhythmia; *i.e.*, that intervals which are obviously long or short by comparison with their fellows may be slightly longer or shorter than they would be were it not for the presence of a retarding or accelerating influence. The experiments here reported were undertaken to determine whether or not in certain cases of auricular fibrillation there are such periodic influences in connection with respiration, and if so, how great is their extent.

The method of investigation was by the use of averages. After recording a long series of respirations synchronously with a pulse curve and time record, each respiratory cycle was divided into eight parts. The pulse intervals were then measured in hundredths of a second and divided into eight groups according to the part of a respiratory cycle in which each happened to end; and finally the numbers in each group were averaged and the averages plotted (the shaded areas in the accompanying figures) under a diagrammatic curve representing respiration. Time is indicated at the bottom of the figures to show the respiratory rates. The figures of each table (which actually apply to one respiratory cycle) are charted over two cycles in order to show more clearly the relations of pulse and respiration in all parts of the cycle.

As was to be expected, the outlines of the shaded areas thus formed are irregular in most cases; and, as a rule, these minor irregularities cannot

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be eliminated without an exceedingly laborious increase in the number of pulse intervals measured and tabulated. Fortunately such a condition is not essential for the purposes of these experiments, which are expected to show only the simplest relationship which may exist between pulse intervals and respiration, such as a tendency for longer intervals on one side of the respiratory cycle than on the other; so that only the larger "waves" in the pulse interval curve are to be considered. In thus limiting the scope of the investigation it is of course recognized that an extension of the analysis to include a sufficient number of measurements might establish the identity of two or more regions in the respiratory cycle where tendencies in varying degrees exist for the production of longer or shorter heart intervals.

In order to show more clearly the large waves in the plotted curve, the irregularities resulting from the original averages have been "smoothed out" by use of the following well recognized method: In each eighth of the respiratory cycle, instead of plotting simply the average of the heart intervals terminating in that period, the average (properly weighted⁶) was plotted for this eighth together with the preceding and the following eighths. Thus, in the revised curve (which is shown in all the figures by the dotted line) each plotted point represents an average of all heart intervals terminating in three-eighths of the respiratory cycle. Thus, if it is desired to contrast inspiration and expiration, the dot in the third respiratory division gives the general average heart interval for the second, third and fourth divisions or the main part of inspiration, and the dot in the seventh space a similar average for the sixth, seventh and eighth divisions or the major part of expiration, and so on.

In cases where all heart beats corresponded to palpable waves at the periphery, the heart rate was taken from impulses in the brachial artery communicated to an elastic pad strapped to the arm. The respiratory movements were recorded by a similar receiver fastened about the chest, and the time was indicated in fifths of a second by a Jaquet time-marker—all registering on a large Hürtle kymograph carrying about three meters of wide smoked paper. About eight or ten minutes' continuous record constituted an experiment. When abortive beats were present the intervals between systoles were measured on the electrocardiographic record with a simultaneous tracing of respiration and time indicated in twenty-fifths of a second. In the absence of a film camera the ordinary 8 × 17 cm. plates were exposed in six divisions, and several plates were used in succession.

The breathing was fairly regular in all cases and only typical respiratory cycles were used in the analyses; *i.e.*, curves disturbed by cough, movements of the body, etc., were thrown out. In dividing the respiratory cycles a mark was placed at the summit of each inspiration (inspiration is represented by the up-stroke of the curve), or where a wave was rounded or elongated, at the midpoint between the inspiratory and expiratory movements as nearly as could be determined by the eye. Another point was similarly marked in the middle of each interval between respirations, and the space

between each "summit" and "trough" was then divided into four equal parts. The divisions of each respiratory cycle were then numbered from one to eight beginning with the space after each "trough" mark. These approximate divisions formed by free-hand measurements are adequate for the needs of the experiment because the final curve is but little affected if a given pulse interval which ends near one of the dividing lines is included in one or the other division. In averages from large numbers misplacements one way are balanced by others in the opposite direction. To rule out a constant error in one direction the important precaution was taken of dividing the respiratory cycles without reference to the pulse waves, the distribution of which might thereby be affected.

The following nine cases of auricular fibrillation were studied:—

Case A. No. 12760. Female. Aged 66 years.

Diagnosis: Cerebral thrombosis, arteriosclerosis, chronic myocarditis with auricular fibrillation and block of right branch of bundle of His.

She had had rheumatic fever at the age of 20 years, and ever since then had been more or less short of breath, but without definite breaks in compensation. Complement fixation for syphilis negative. She was in the hospital

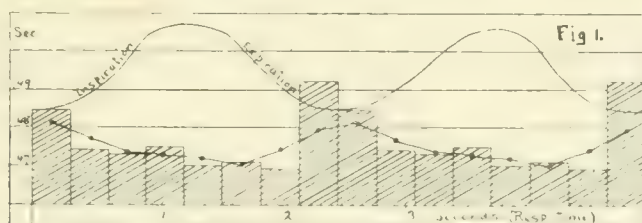


Fig. 1. Case A. No decided relation of pulse intervals to respiration.

for hemiplegia, and was gradually regaining function when this experiment was made. She had no heart symptoms and had not been taking digitalis. Electrocardiograms during normal breathing, without drugs, showed auricular fibrillation, right branch block and occasional ectopic beats. The latter were omitted from the tabulation of heart intervals which follows:—

Av. of 84 heart intervals ending in	1st div. of resp. cycles	=	.485 sec.
" 79 " " "	2nd " " "	=	.474 "
" 90 " " "	3rd " " "	=	.473 "
" 72 " " "	4th " " "	=	.495 "
" 93 " " "	5th " " "	=	.470 "
" 93 " " "	6th " " "	=	.471 "
" 91 " " "	7th " " "	=	.469 "
" 78 " " "	8th " " "	=	.482 "
<hr/>			
680 = total.			

These averages are shown graphically in Fig. 1. There is an apparent tendency for longer intervals between inspiration and expiration, but the amount of irregularity is such that as an isolated experiment it would have to be considered inconclusive.

Case B. No. 10089. Male. Aged 48 years.

Diagnosis: Mitral stenosis and insufficiency, tricuspid and pulmonary insufficiency, auricular fibrillation.

He had had frequent tonsillitis, rheumatic fever at the age of 7 and again seven years ago. Venereal disease was denied and the complement fixation test was negative. The heart condition has changed little in the three or four years during which the patient has been under observation. On the day of admission to the hospital when many abortive beats were present, electrocardiograms were taken, which showed typical auricular fibrillation. Later, when all beats could be registered at the periphery, many more measurements were made by the use of the kymograph. At all times the patient was under the influence of digitalis—about 1 c.c. or more of the tincture daily.

The original electrocardiograms (October the 28th, 1915) provided 162 pulse-interval measurements. The results are shown in the following averages and in Fig. 2.

Av. of 21 heart intervals ending in	1st div. of resp. cycles	=	.622 sec.
" 16 "	2nd "	=	.569 "
" 21 "	3rd "	=	.636 "
" 22 "	4th "	=	.629 "
" 19 "	5th "	=	.600 "
" 23 "	6th "	=	.607 "
" 16 "	7th "	=	.634 "
" 24 "	8th "	=	.635 "
162	total.		

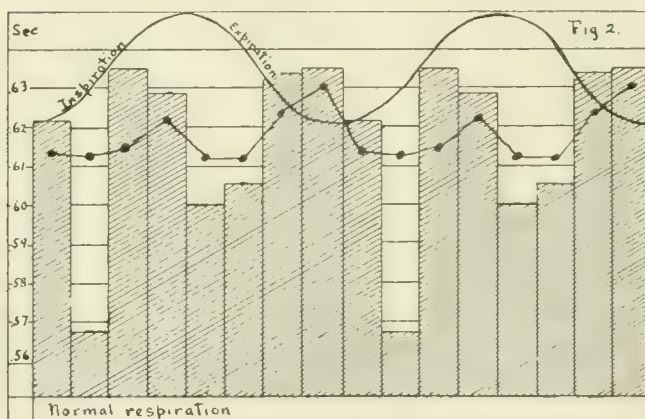


Fig. 2. *Case B.* From entrance electrocardiograms.

As was later discovered, the number of intervals included in this tabulation was quite inadequate to give consistent results, so that no deductions can be drawn from it either for or against a relationship between pulse intervals and respiration.

After eight days' rest he had become considerably improved and palpable pulse waves corresponded to all heart beats. Kymographic records were then obtained with the patient half reclining during quiet and during forced breathing, both before and after the administration of atropin.

Analysis of the "quiet breathing without atropin" tracings resulted as follows :—

Av. of 105 heart intervals ending in	1st div. of resp. cycles	=	.820 sec.
" 68 " " "	2nd " " "	=	.807 "
" 132 " " "	3rd " " "	=	.797 "
" 63 " " "	4th " " "	=	.870 "
" 102 " " "	5th " " "	=	.783 "
" 114 " " "	6th " " "	=	.810 "
" 75 " " "	7th " " "	=	.821 "
" 102 " " "	8th " " "	=	.780 "
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761	total.		

During this time the respiration rate was 26 and the pulse rate 74. The results of the experiment, as displayed in Fig. 3, are again too irregular for definite conclusions; though there is a suggestion of shorter intervals between respiratory waves.

In the next experiment, which followed immediately, the breathing was regulated so as to be deep and fairly regular at the rate of 10 per minute: pulse rate was 76. Analysis of the results follows :—

Av. of 71 heart intervals ending in	1st div. of resp. cycles	=	.780 sec.
" 80 " " "	2nd " " "	=	.808 "
" 81 " " "	3rd " " "	=	.786 "
" 79 " " "	4th " " "	=	.775 "
" 85 " " "	5th " " "	=	.780 "
" 91 " " "	6th " " "	=	.790 "
" 93 " " "	7th " " "	=	.816 "
" 88 " " "	8th " " "	=	.796 "
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668	= total.		

These results, which are shown graphically in Fig. 4, are again quite irregular.

Following these two tracings the patient was given atropin hypodermically .001 (1.50 mg. per kilo of body weight) and fifteen minutes later the "quiet breathing" experiment was repeated. Respiration was now at the rate of 28 and pulse 93 per minute. Analysis of the results was as follows :—

Av. of 101 heart intervals ending in	1st div. of resp. cycles	=	.647 sec.
" 110 " " "	2nd " " "	=	.618 "
" 160 " " "	3rd " " "	=	.656 "
" 72 " " "	4th " " "	=	.673 "
" 161 " " "	5th " " "	=	.622 "
" 114 " " "	6th " " "	=	.672 "
" 97 " " "	7th " " "	=	.614 "
" 162 " " "	8th " " "	=	.640 "
<hr/>			
977	total.		

These averages, as shown by the graph in Fig. 5, are quite irregular and inconclusive, though they agree with the other "normal breathing"

experiment (Fig. 3) in suggesting a tendency for shorter intervals between the respiratory acts.

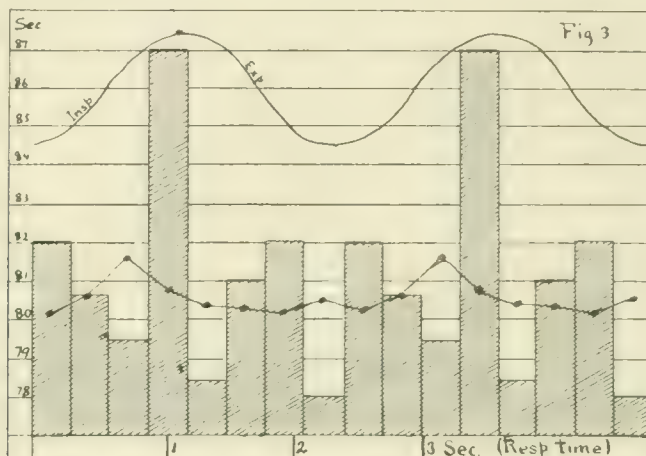


Fig. 3. Case B. A few days later. Normal breathing, no atropin.

Thirty minutes after the atropin injection the "forced breathing" experiment was repeated. As before, respirations were deep and slow at the rate of 10 per minute, pulse rate about 99. Analysis of this tracing was as follows:—

Av. of 83 heart intervals ending in	1st div. of resp. cycles	=	.604 sec.
" 74 " " "	2nd " " "	=	.607 "
" 81 " " "	3rd " " "	=	.592 "
" 77 " " "	4th " " "	=	.597 "
" 89 " " "	5th " " "	=	.608 "
" 89 " " "	6th " " "	=	.606 "
" 80 " " "	7th " " "	=	.611 "
" 84 " " "	8th " " "	=	.603 "

657 = total.

As shown in Fig. 6 there is again no certain relation to be seen between respiration and pulse intervals, although here again there is a suggestive resemblance to the other "forced breathing" experiment (Fig. 4).

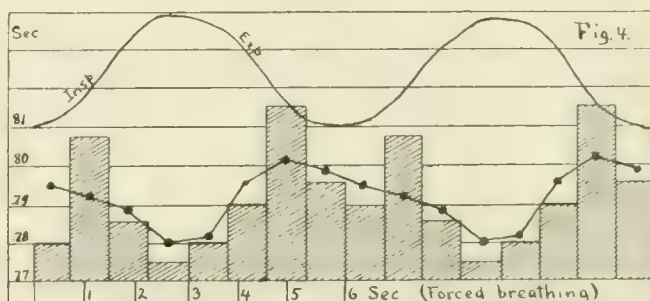


Fig. 4. Case B. Forced breathing, without atropin.

A year later the same patient while again taking digitalis and during a period of fair compensation was re-examined electrocardiographically and the same irregular results obtained as shown in the following table and Fig. 7.

Av. of 32 heart intervals ending in	1st div. of resp. cycles		.899 sec.
" 58 "	" 2nd "	" "	.948 "
" 30 "	" 3rd "	" "	.910 "
" 27 "	" 4th "	" "	.939 "
" 23 "	" 5th "	" "	.818 "
" 67 "	" 6th "	" "	.935 "
" 29 "	" 7th "	" "	.963 "
" 28 "	" 8th "	" "	.855 "
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294	— total.		

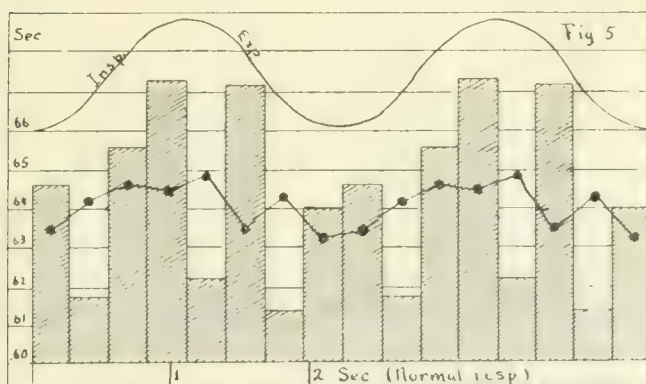


Fig. 5. Case B. Normal breathing, after atropin injection.

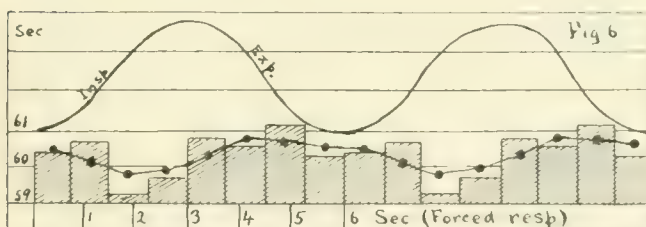


Fig. 6. Case B. Forced breathing, after atropin injection.

All six experiments on this patient, therefore, must be regarded as having failed to show any consistent relationship between respiratory phases and heart intervals. In fact, if it were not for the suggested agreement between the two "normal breathing" experiments (Figs. 3 and 5) and between the two "forced breathing" experiments (Figs. 4 and 6), they would constitute strong presumptive evidence that there was no such relationship.

Case C. No. 9912. Female. Aged 37 years.

Diagnosis: Mitral stenosis and auricular fibrillation, the latter proved by electrocardiogram.

In addition to frequent attacks of tonsillitis she had had rheumatic fever at the age of 18 and again at 29 years, pneumonia three years ago, "grippe" one year ago. Heart trouble had been recognized for a number of years, and her first decompensation appears to have occurred five years ago.

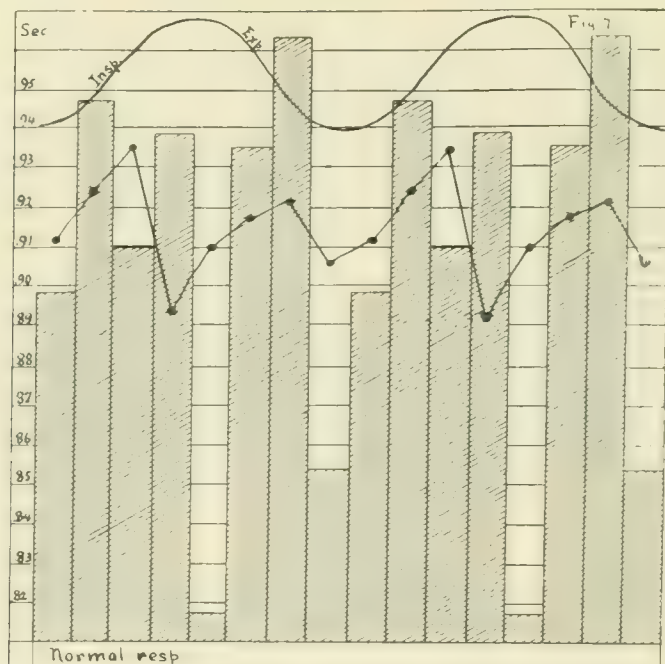


Fig. 7. *Case B. One year later. Normal breathing.*

No evidence of venereal disease. Complement fixation for syphilis negative. The experiments here reported were made during the latter part of the patient's stay in the hospital after several weeks of practically continuous digitalis therapy (daily 4 to 6 c.c. of a standardized tincture), during which there was marked diuresis with disappearance of œdema and reduction of tachycardia. All heart beats could then be registered from the brachial artery, and the kymograph was used.

On October 6th, 1915 (three weeks after entrance), two experiments were made, one before and the other after the administration of atropin. In both the patient was resting quietly in a half reclining position, breathing at the rate of 30 per minute and with a pulse rate of 88 (before) to 90 (after

atropin). The first tracing provided 513 pulse intervals suitable for measurement and classification. This classification resulted as follows:—

Av. of 71 heart intervals ending in				1st div. of resp. cycles.		
..	67	2nd
..	61	3rd
..	63	4th
..	69	5th
..	70	6th
..	59	7th
..	53	8th
513 — total.						

These averages are shown graphically in Fig. 8.

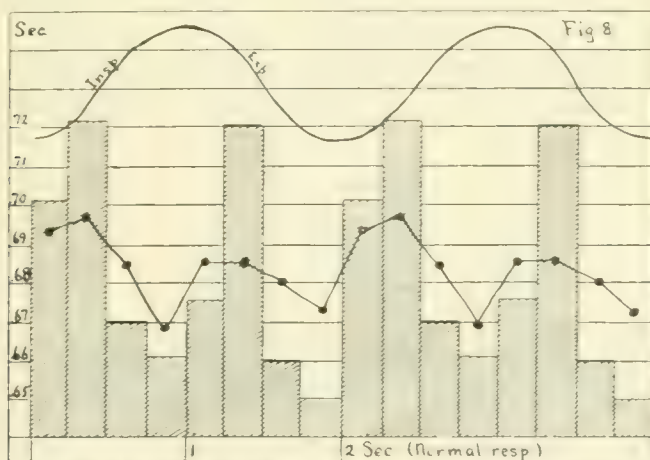


Fig. 8. Case C. Normal breathing, without atropin.

Twenty-five minutes after a subcutaneous injection of .00114 gm. atropin (1/50 mgm. per kilo body weight) the second tracing was taken. The results of this analysis (see Fig. 9) were as follows:—

Av. of 77 heart intervals ending in				1st div. of resp. cycles		
..	76	2nd
..	66	3rd
..	63	4th
..	80	5th
..	61	6th
..	94	7th
..	48	8th
565 — total						

As will be seen from Figs. 8 and 9, both of these experiments produced irregular results, so that deductions cannot be drawn from them in spite of the curious similarity between the dotted graphs representing the corrected averages. On account of the variability of pulse intervals in this case (from .460 to 1.350 second) several thousand measurements might be required to obtain a smooth curve.

Three weeks later two more records were obtained from this case, one during quiet breathing at the rate of 22 per minute, the other during slow,

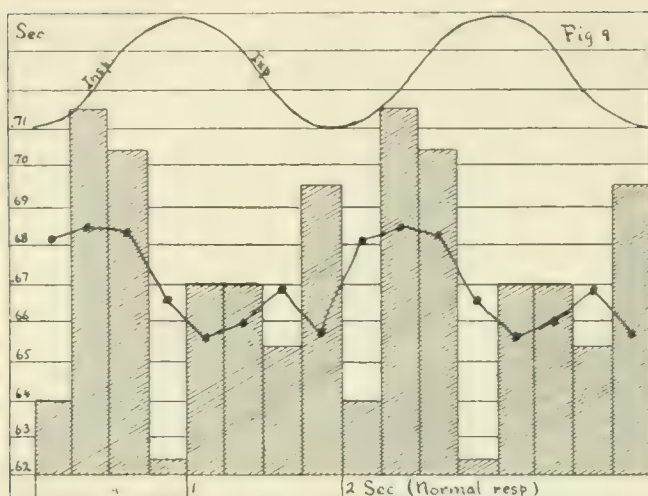


Fig. 9. Case C. Normal breathing, with atropin.

deep, "forced" respirations at the rate of 6 per minute. The pulse at this time was very much slower, averaging 45 per minute during the quiet breathing and 55 per minute during the forced respiration. The variability of the intervals also was less at this time (.72 to 1.45 second during quiet breathing, and .88 to 1.60 second during the forced breathing). Analysis of the "ordinary respiration" tracing resulted as follows:—

Av. of 51 heart intervals ending in	1st div. of resp. cycles	=	1.320 sec.
" 47 "	" 2nd "	"	1.337 "
" 40 "	" 3rd "	"	1.321 "
" 44 "	" 4th "	"	1.327 "
" 49 "	" 5th "	"	1.320 "
" 38 "	" 6th "	"	1.360 "
" 42 "	" 7th "	"	1.331 "
" 39 "	" 8th "	"	1.352 "

350 = total.

These averages are shown graphically in Fig. 10, which indicates a rather definite though slight shortening of the intervals ending during inspiration and a tendency to lengthen during expiration.

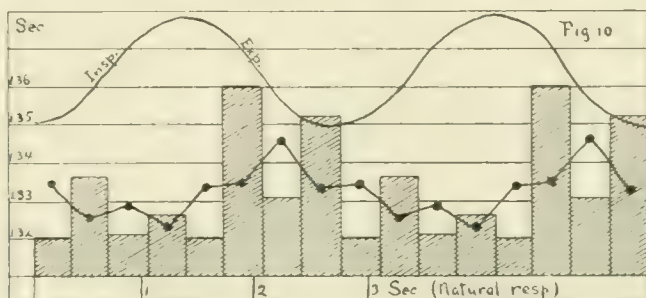


Fig. 10. Case C. Later with slower rate. Normal breathing, no atropin

Fig. 11 shows that on the "forced breathing" curve the effect of respiration is in the same direction and is very prominent, thus:—

Av. of 58 heart intervals ending in	1st div. of resp. cycles	=	1.16	sec.
.. 48	2nd	=	1.055	..
.. 50	3rd	=	1.110	..
.. 47	4th	=	1.220	..
.. 47	5th	=	1.247	..
.. 44	6th	=	1.310	..
.. 39	7th	=	1.190	..
.. 48	8th	=	1.218	..
381	total.			

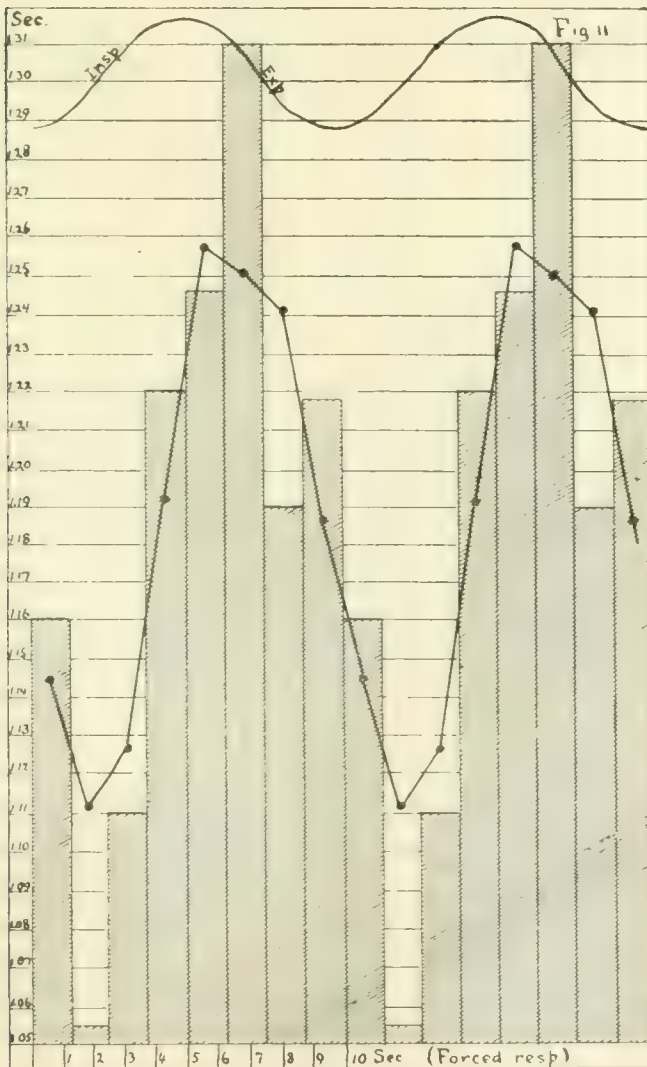


Fig. 11. Case C. Same with forced breathing.

Case D. No. 10079. Male. Aged 64 years.

Diagnosis: Myocarditis, auricular fibrillation, hydrothorax. The patient had had mumps, whooping cough and diphtheria in childhood, a severe attack of measles at the age of 26, and rather frequently tonsillitis until about fifteen years ago; pleurisy three or four years ago. Venereal disease was denied and complement fixation test negative. He has known that his heart was irregular during the last twenty years. The examinations on this patient were made after three or four weeks of rest in the hospital. On account of the continued presence of abortive beats, however, it was necessary for measuring the heart rate to use the electrocardiograph; the patient was sitting. In addition to the auricular fibrillation, the heart action was complicated by the frequent appearance of abnormal ventricular contractions which arose from at least three different foci. Three or four c.c.

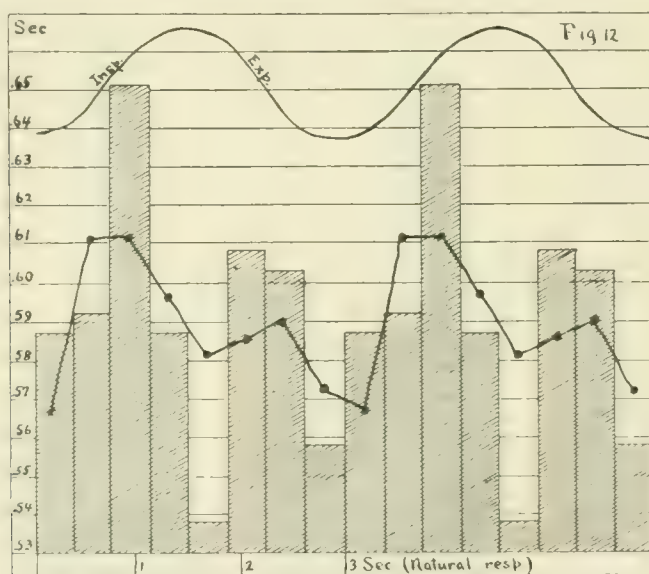


Fig. 12. Case D. Normal breathing, without atropin.

of tincture of digitalis daily had been administered for several weeks, but had been discontinued nine days before these experiments. Records were taken first during quiet breathing, then during forced breathing, and again during forced breathing after atropin injection.

During the first observation the heart rate was 101, respiration rate 20. The results were as follows:—

Av. of 27 heart intervals ending in	1st div. of resp. cycles	=	.588 sec.
.. 31	2nd	=	.592 ..
.. 34	3rd	=	.651 ..
.. 34	4th	=	.587 ..
.. 25	5th	=	.538 ..
.. 32	6th	=	.608 ..
.. 32	7th	=	.603 ..
.. 31	8th	=	.558 ..

246 = total.

These averages, as shown in Fig. 12, are too irregular to be of much value, but suggest a tendency for shorter intervals in expiration.

During the next observation the patient breathed slowly and deeply about eleven times per minute and the pulse rate was 120. The following is the analysis of this experiment :—

Av. of 28 heart intervals ending in	1st div. of resp. cycles	=	.452 sec.
.. 17	2nd	=	.481 ..
.. 28	3rd	=	.530 ..
.. 26	4th	=	.558 ..
.. 25	5th	=	.488 ..
.. 32	6th	=	.495 ..
.. 32	7th	=	.530 ..
.. 31	8th	=	.457 ..
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219	= total.		

The resulting curve (Fig. 13), though somewhat irregular, again suggests a tendency for longer intervals to occur during inspiration and shorter ones during expiration.

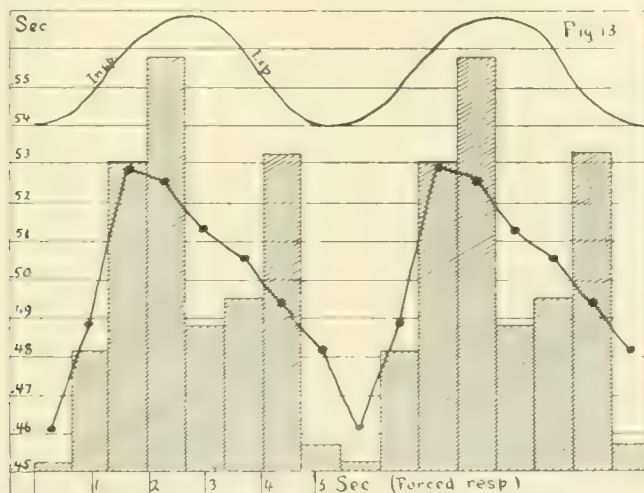


Fig. 13. Case D. Forced breathing, without atropin.

At the same sitting the "forced breathing" experiment was repeated about fifteen minutes after a subcutaneous administration of one milligram of atropin (1/50 mg. per kilo body weight). Respirations as before, heart rate 162. This experiment yielded the following results :—

Av. of 48 heart intervals ending in	1st div. of resp. cycles	=	.371 sec.
.. 43	2nd	=	.397 ..
.. 44	3rd	=	.369 ..
.. 39	4th	=	.386 ..
.. 40	5th	=	.372 ..
.. 40	6th	=	.372 ..
.. 48	7th	=	.372 ..
.. 47	8th	=	.357 ..
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349	= total.		

The graph formed by these averages (Fig. 14) is again somewhat irregular, but the suggestion it offers is again in harmony with the two preceding experiments on the same case. Taken separately, Figs. 12, 13 and 14 are quite inconclusive: considered together, they suggest very strongly a relation between heart rhythm and respiration.

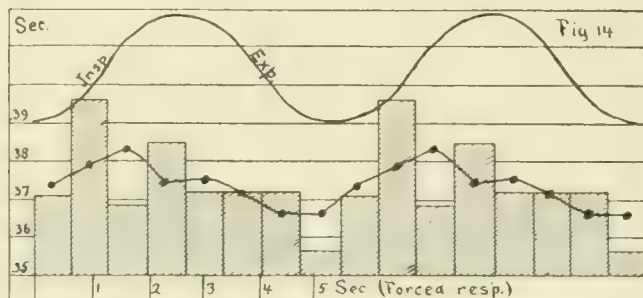


Fig. 14. Case D. Forced breathing, with atropin.

Case E. No. 9647. Male. Aged 64 years.

Seen by the courtesy of the Surgical Service, to which he was admitted for fracture of the femur. The heart diagnosis without graphic study was auricular fibrillation without valvular disease. The patient had had measles, mumps, scarlet fever in childhood: typhoid fever at the age of 42, pneumonia at the age of 55. He denied venereal disease. He had known that his heart was irregular since his pneumonia nine years ago, though there had never been outspoken decompensation. He had not taken digitalis.

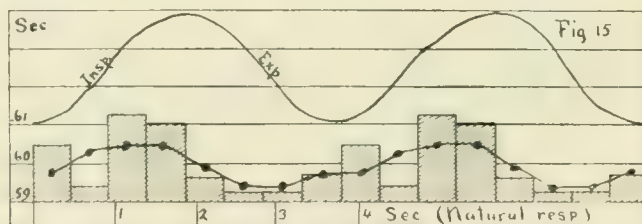


Fig. 15. Case E. Normal breathing. No drugs.

Kymographic tracings were taken while the patient was lying in bed with respiration rate 16 and pulse rate 100. The following is the analysis of this tracing:—

Average of 38 heart intervals ending in	1st div. of resp. cycles	=	.605 sec.
.. 43	2nd	=	.594 ..
.. 40	3rd	=	.612 ..
.. 36	4th	=	.610 ..
.. 43	5th	=	.597 ..
.. 37	6th	=	.592 ..
.. 36	7th	=	.592 ..
.. 39	8th	=	.598 ..

312 — total.

These averages, as shown in Fig. 15, have a tendency for longer intervals during inspiration and shorter ones during expiration—almost the exact counterpart of Fig. 14.

Case F. No. 12614. Male. Aged 34.

Diagnosis, established by electrocardiograms and subsequent autopsy: Auricular fibrillation, chronic fibrous myocarditis, syphilitic aortitis (complement fixation was positive), mitral, aortic, and tricuspid disease. He had had gonorrhœa *aet.* 17, chancre *aet.* 18, and "rheumatism" (type not clear) *aet.* 29. There was recurrent ascites, and during life Pick's disease was suspected. During both the following experiments the patient was in partial compensation, half reclining; and, as no abortive beats were present, pulse tracings were used. The first tracing was taken November the 14th 1916, during normal breathing, the second November the 20th, during slow forced breathing at the rate of about 9 per minute. Four c.c. daily of the tincture of digitalis was taken from November the 9th to 16th, diuretin 4 gms. daily November the 16th to 24th.

The following table shows the tabulation of the "natural breathing" experiment:—

Av. of 102 heart intervals ending in	1st div. of resp. cycles	=	.770 sec.
" 221 "	2nd "	=	.807 "
" 90 "	3rd "	=	.851 "
" 171 "	4th "	=	.790 "
" 187 "	5th "	=	.826 "
" 119 "	6th "	=	.772 "
" 206 "	7th "	=	.793 "
" 140 "	8th "	=	.790 "

1,236 -- total.

As shown by Fig. 16, these results show a strong resemblance to the two foregoing cases.

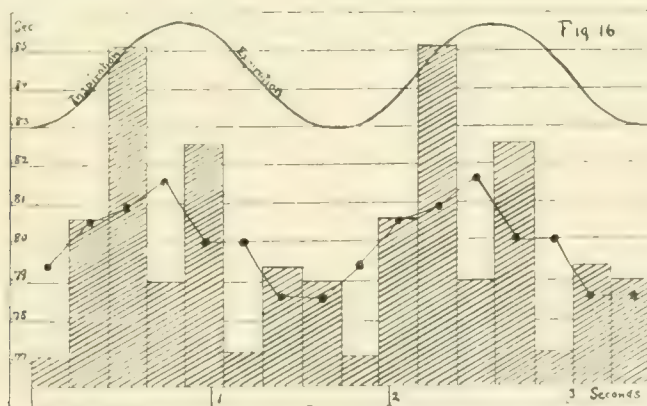


Fig. 16. Case F. Normal breathing, no atropin.

The "forced respiration" experiment, which is exhibited in the following table and in Fig. 17, produced quite irregular results; but the slight tendency

to periodicity which it does show is roughly in harmony with the first experiment.

Av. of 202 heart intervals ending in	1st div. of resp. cycles	=	.731 sec.
" 184 "	" 2nd "	=	.727 "
" 217 "	" 3rd "	=	.731 "
" 212 "	" 4th "	=	.738 "
" 212 "	" 5th "	=	.721 "
" 198 "	" 6th "	=	.729 "
" 190 "	" 7th "	=	.720 "
" 190 "	" 8th "	=	.730 "
1,605 = total.			

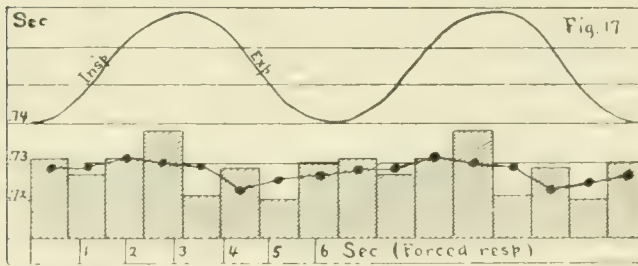


Fig. 17. Case F. Forced breathing, no atropin.

Case G. No. 12771. Male. Aged 57.

Diagnosis: Aortic and mitral insufficiency, mitral stenosis, auricular fibrillation. The origin of his infection was not known—possibly oral sepsis. No history of rheumatism or syphilis. Complement fixation negative. At the time of these experiments he was in fairly good compensation and had taken no digitalis for four or five weeks previously. There being no abortive beats, pulse tracings were used for both experiments, the first on December the 6th, 1916, during natural breathing, and the second December the 17th, during deep respiration.

The "natural breathing" experiment resulted as follows:—

Av. of 233 heart intervals ending in	1st div. of resp. cycles	=	.719 sec.
" 221 "	" 2nd "	=	.732 "
" 185 "	" 3rd "	=	.739 "
" 224 "	" 4th "	=	.734 "
" 197 "	" 5th "	=	.766 "
" 241 "	" 6th "	=	.712 "
" 234 "	" 7th "	=	.738 "
" 204 "	" 8th "	=	.715 "
1,739 = total.			

The corresponding graphic representation (Fig. 18) shows a remarkable similarity to the foregoing figures. The same tendency to shorter intervals

between respiratory acts appears in the "forced breathing" experiment as shown in the following table and in Fig. 19.

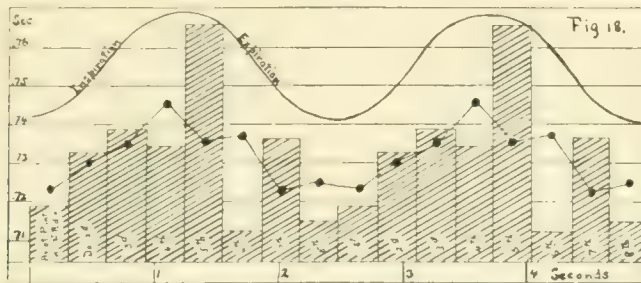


Fig. 18. Case G. Normal breathing, no atropin.

Av. of 199 heart intervals ending in	1st div. of resp. cycles	=	.671 sec.
" 192 "	2nd "	"	= .677 "
" 216 "	3rd "	"	= .664 "
" 201 "	4th "	"	= .678 "
" 212 "	5th "	"	= .689 "
" 214 "	6th "	"	= .674 "
" 198 "	7th "	"	= .673 "
" 200 "	8th "	"	= .665 "

1,632 = total.

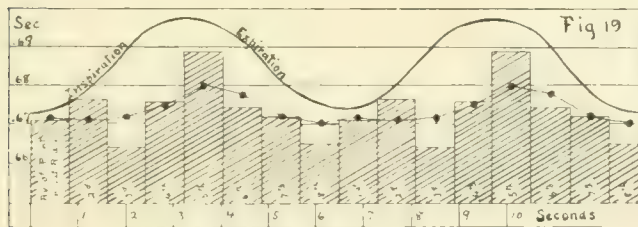


Fig. 19. Case G. Forced breathing, no atropin.

Case H. No. 12739. Female. Aged 50.

Diagnosis: Hyperthyroidism, probably also with some thymic disturbance. The acute history was a matter of two or three months previous to these experiments (November the 29th, 1916). No definite history of infections. Complement fixation negative. She had had a little digitalis three or four weeks before. The auricular fibrillation was paroxysmal in character, alternating every few days with periods of normal heart beats with a rate of about 90. A series of electrocardiograph tracings during one of the paroxysms yielded the results indicated in the following table and in Fig. 20. The striking similarity to preceding figures will be observed.

Av. of 100 pulse intervals ending in	1st div. of resp. cycles	=	376 sec.
" 96 " " "	2nd " " "	=	376 "
" 121 " " "	3rd " " "	=	376 "
" 123 " " "	4th " " "	=	404 "
" 105 " " "	5th " " "	=	386 "
" 104 " " "	6th " " "	=	395 "
" 114 " " "	7th " " "	=	374 "
" 112 " " "	8th " " "	=	372 "
875 = total.			

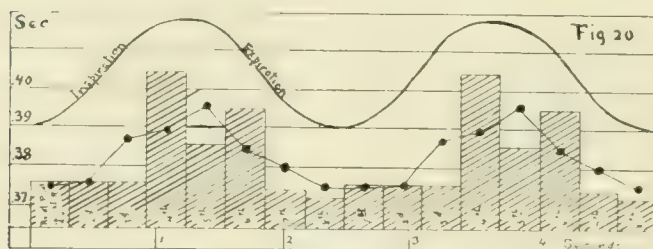


Fig. 20. Case H. Normal breathing, no atropin.

Case I. No. 9371. Male. Aged 38 years.

Diagnosis: Exophthalmic goitre of thyroid and thymic type, acute and chronic infected tonsils, auricular fibrillation—the latter indicated by typical electrocardiograms. The patient had had mumps, measles, whooping cough and varicella in early life; there was no evidence of venereal disease. Symptoms of hyperthyroidism have been pronounced during the last three years, and exophthalmos became very marked. There were many abortive heart beats during the early part of the patient's stay in the hospital; but the experiments here reported were made later after three weeks' continuous digitalis medication (3 c.c. of the tincture daily) when all beats could be registered from the arm by means of the kymograph. The patient was in the horizontal position in all of the three experiments.

On September the 22nd, 1915, during normal respiration at the rate of 10 per minute and pulse rate 90, a tracing was taken which resulted in the following analysis:—

Av. of 82 heart intervals ending in	1st div. of resp. cycles	=	618 sec.
" 77 " " "	2nd " " "	=	668 "
" 77 " " "	3rd " " "	=	664 "
" 69 " " "	4th " " "	=	676 "
" 86 " " "	5th " " "	=	670 "
" 83 " " "	6th " " "	=	658 "
" 97 " " "	7th " " "	=	642 "
" 81 " " "	8th " " "	=	628 "
652 = total.			

As exhibited in Fig. 21 these averages show a convincing relation to respiration as in the last several cases described, the shorter intervals tending to occur during expiration and the longer ones during inspiration.

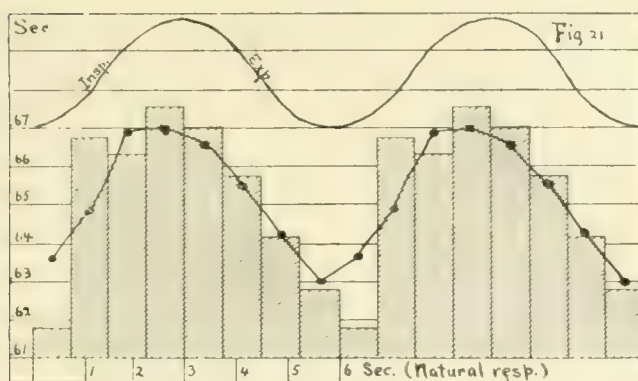


Fig. 21. Case I. Normal breathing, no atropin.

On September the 24th the experiment was repeated on this case fifteen minutes after the subcutaneous administration of half milligram of atropin (1/100 mg. per kilo body weight). Respiration was again 10 per minute, pulse 102. The following analysis was made of the tracing:—

Av. of 124 heart intervals ending in	1st div. of resp. cycles	=	.523 sec.
" 103 "	2nd "	=	.591 "
" 105 "	3rd "	=	.582 "
" 102 "	4th "	=	.585 "
" 117 "	5th "	=	.603 "
" 109 "	6th "	=	.588 "
" 111 "	7th "	=	.572 "
" 114 "	8th "	=	.556 "

885 = total.

These results, as exhibited graphically in Fig. 22, again show striking agreement with preceding curves.



Fig. 22. Case I. Normal breathing, with small amount of atropin.

On the following day the experiment was again repeated fifteen minutes after another subcutaneous administration of atropin. At this time, however, double the former dose was given ($\cdot 001$ or $1/50$ mg. per kilo body weight). Respiration was now 12 per minute, pulse 112. The following analysis resulted:—

Av. of 118 heart intervals ending in	1st div. of resp. cycles	—	$\cdot 522$ sec.
.. 103	2nd	—	$\cdot 523$..
.. 105	3rd	—	$\cdot 545$..
.. 104	4th	—	$\cdot 536$..
.. 115	5th	—	$\cdot 546$..
.. 107	6th	—	$\cdot 550$..
.. 113	7th	—	$\cdot 540$..
.. 114	8th	—	$\cdot 522$..
879	total.		

As shown in Fig. 23, the results differ only in degree (the relation to respiration is a little less marked) from those in the other two experiments.

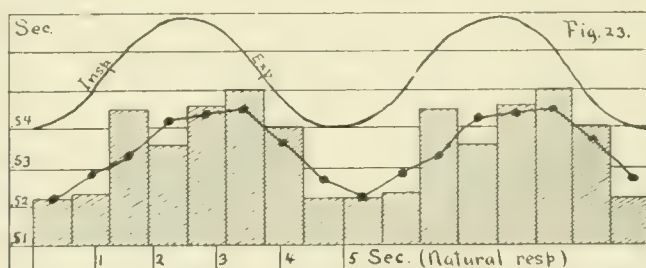


Fig. 23. Case I. Normal breathing, with larger dose of atropin.

Summary and Discussion.

Out of nine cases of auricular fibrillation studied, six (D, E, F, G, H and I) show a fairly consistent tendency for shorter heart intervals during late expiration or early inspiration than during the opposite part of the respiratory cycle. This is shown by the figures for the combined averages (dotted lines) of Figs. 12 to 23 inclusive. Singly, several of these cases would give reason for seriously questioning the relationship to respiration, since there is much variation in some of the original averages (shaded areas); but being in close agreement and constituting two-thirds of the cases studied, the suggestion they offer is very convincing. It is rendered very much more convincing, however, by the fact that in the four of these six cases in which repeated experiments were made they all yielded corresponding results. The last case is the most striking, showing on each of three occasions results so nearly alike that the plotted curves might be almost exactly superposed. The slight quantitative divergence noticeable in the last experiment (Fig. 23) may be related to the increased dose of atropin. The similarity between the two cases of hyperthyroidism is shown by comparing the last trio with Fig. 20.

Of the three remaining cases studied, one (*Case C*) also shows a highly probable relation between pulse intervals and respiratory phases, but was excluded from the foregoing group for the reason that the results of all four experiments on this case do not agree. As a matter of fact, however, in the interim between the first two and the last two observations on this case there was a great change, under digitalis the pulse rate falling from 88 to 45 per minute. With such an alteration in rate it is not surprising to find a difference in the pulse relation to respiration. It is specially to be noted that the results of the first two experiments and also of the second two agree closely with each other. The first two (Figs. 8 and 9) suggest a complex relation to respiration, *i.e.*, a tendency to acceleration of the heart at more than one part of the respiratory cycle. Such a finding could not be substantiated, however, without a very large series of measurements; and all that can be said here is that no definite relation or lack of relation is proved by Figs. 8 and 9. Their resemblance may be coincidental. Analyses made at the lower rate (Figs. 10 and 11) both show a tendency to shorter pulse intervals in early inspiration—only slightly later than in most of the six cases described above. Fig. 12 by itself with its wide swinging and regular curve would be a remarkable coincidence if the relation to respiration there shown were fortuitous—and much the more so when it is seen that Figs. 11 and 12 differ only in degree, that the greater apparent effect was obtained during forced breathing, and that the resemblance to the six following cases is close.

No consistent type of curve appears among the six experiments on *Case B*. There is a suggestive similarity between the two "normal breathing" curves (Figs. 3 and 5, which also resemble the curves from *Cases D, E, F, G, H and I*) and the two "forced breathing" curves, which show an opposite tendency. But taken as a whole, the six curves from this case rather suggest an absence of relationship between respiration and pulse intervals. To *prove* the absence of such relationship would, of course, require an exceedingly large series of measurements.

Similarly, the one experiment for *Case A* (Fig. 1) must be considered inconclusive, but suggesting an absence of relationship between respiration and heart intervals.

The selection of the end of each pulse interval as the criterion for deciding its place in the respiratory cycle is not entirely arbitrary. It was chosen because it is the time when conditions combine to determine the duration of the interval, and the object was to find out what part respiration plays among these determining conditions. If another scheme of classification had been used, *e.g.*, if a given pulse interval were counted as belonging to a certain eighth of a respiratory cycle because it happened to begin there or because its midpoint was there, the resulting curves would be correspondingly shifted to the left in relation to the respiration diagrams (by about one average pulse interval in the one case and by half that time in the other). The curves would also be altered in shape, but only so in

minor details. Comparisons have been made in several cases and the distinctive features of the curves, except for their displacement to the left, were the same, whichever method of classification was used.

Another possible variation in the method of analysis would be to substitute for the arithmetic mean of each group of pulse intervals the median.* This also has been tried and was found to make slight alterations in the shape of the resulting curves but not to alter their main characteristics.

Interesting questions concerning its mechanism are raised by the finding of a respiratory effect on the heart rate in the presence of auricular fibrillation. The first is the relation of this effect to the ordinary respiratory arrhythmia of hearts which beat normally. In these non-fibrillating cases the fact that the place of origin and the conduction paths of the heart beat remain unaltered is conclusive evidence that the retarding or accelerating influence acts through the sinus; and the term sinus arrhythmia is therefore appropriate. That the influence is neurogenic is a natural supposition, and is corroborated by the fact that it is reduced or eliminated by atropin,² or by vagal section in animals.

It is equally clear that the retarding or accelerating respiratory influences in the fibrillating cases have a different mechanism; for present knowledge of the heart beat makes highly improbable a sinus influence on the ventricular contractions in the presence of auricular fibrillation. Evidently periodic influences connected with respiration may take effect on a lower part of the heart. Further than this the discussion of the mechanism becomes frankly speculative.

It is possible that periodic changes in the force or frequency or other characteristics of the auricular fibrillary contractions may modify the time of the ventricular responses. That differences in effectiveness exist among the impulses passing from the fibrillating auricle would seem highly probable from the irregularity in the ventricular responses. And at least a difference between strength of impulses of sinus origin and those from fibrillary contractions may be suspected from the occasional observation of a fall of ventricular rate coincident with the onset of clinical fibrillation. It is also probable that the character of auricular fibrillary contractions may be modified by nerve impulses, since in cases in which the characteristic fine diastolic tremor of the galvanometer string is not at first apparent, these electric oscillations may be seen after vagus stimulation. Also experimental fibrillation may change from fine to coarse or *vice versa* during the progress of an experiment.³ So far as I am aware, however, phasic variations of this character corresponding to respirations have not been observed. They did not appear in the electrocardiograms of these cases. And if they were observed, proof would still be required that they were associated with corresponding differences in intensity of the impulses to the ventricle.

* The "median" in a series of variables is the value above which and below which occur equal numbers of the variable.⁶

A second possibility is that variations occur in the rate of recovery of excitability in the ventricle. This excitability being one of the governing factors which decide the effect or non-effect of auricular impulses which force their way through the conducting bundle, it is easy to see that if it rises more gradually in one phase of respiration than another the pulse intervals during that phase will be found to have a longer average, notwithstanding the fact that frequently short pulse intervals occur in this phase when stronger auricular impulses are able to take effect in spite of the lower excitability.

The same may be said in reference to conductivity, which is well known to be subject to nervous influence, and which if alternately increased and diminished would correspondingly increase and diminish the chances for prompt impulse transmission and early ventricular excitation. Change in conductivity has been observed to accompany change of the sinus rate⁴; and in experimental auricular fibrillation reduction of ventricular rate through vagus stimulation has been shown to be associated with lowered bundle conductivity—a lengthened *a-c* interval being demonstrated when the auricle suddenly resumed its regular action⁵. Respiratory fluctuations in the length of the *P-R* interval, so far as I know, however, have not been described.

The respiratory influence on the heart rate, it will be remembered, could not be clearly traced in *Case C* until the pulse rate had become very slow. It is easy to imagine that with multitudes of auricular impulses pressing for admittance to the ventricle, if the door is nearly closed against them (low conductivity) slight changes in the aperture (fluctuations in conductivity) will affect markedly the ability of these impulses to excite the ventricle; while if the door is comparatively wide open their ability to take effect on the ventricle will depend mainly on its recovery from the refractory state and but little on small changes in the opening of the door.

There is of course nothing antagonistic between the several possible explanations offered. In fact it is reasonable to suppose that if fluctuations in excitability occurred through vagus stimulation, the periods of depressed excitability would be accompanied by lowered conductivity from the same cause; and both these factors would work together to increase the length of the pulse intervals.

All the cases examined except *Cases A* and *E* were at the time or had been within a few weeks under the influence of digitalis, and this may have had its effect on the phenomena observed. The well-marked acceleration following atropin injection in *Case I* may possibly be looked upon as due to a previous vagus tonicité somewhat heightened by digitalis. The lessening of the periodic retarding and accelerating influence after atropin may be attributed to the acceleration rather than to the vagus depression *per se*. However, atropin acceleration of the heart rate in the presence of auricular fibrillation must itself be explained in terms of effects produced upon irritability, conductivity or possibly the character of the auricular fibrillary

contractions, *i.e.*, it cannot be a sinus effect and must be explained in some such way as the respiratory rate variation itself.

The slight lack of uniformity in the results of the experiments, *i.e.*, the fact that in one case the longer pulse intervals tend to occur in one respiratory phase and at a slightly different part of the cycle in another case, is not inconsistent with any of the explanations offered and does not imply a difference in the mechanism. Such variations can be accounted for readily by slight differences in the latent period, which is well known to exist in the action of both the cardiac accelerators and inhibitors. It will be noticed that the tendency to acceleration during late expiration and inspiration is the reverse of the customary relation to respiration in cases of sinus arrhythmia.

The respiratory effects on the average pulse rate as shown by the averages in *Cases C and I* is much greater than the usual respiratory effects on the pulse rate in normal middle-aged individuals.

CONCLUSIONS.

Cases of auricular fibrillation may have superposed upon their apparently absolute arrhythmia periodic tendencies to acceleration and retardation which keep time with respiration. The effect is more marked during slow heart action. It is probably best explainable by neurogenic fluctuations in bundle conductivity or in the rate of ventricular recovery of excitability or both. Periodic changes in the auricular fibrillary contractions may be a factor. Digitalis may have influenced the results in all but two cases examined.

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FURTHER REMARKS ON VENTRICULAR EXTRASYSTOLES AND FIBRILLATION UNDER CHLOROFORM.

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School.**)

SINCE the publication of former papers^{1 2 3} on this subject further experiment has made it evident that the term "light chloroform anæsthesia" requires closer definition in its relation to ventricular extrasystoles and fibrillation. The results in the papers referred to were obtained by placing an animal fully, but not deeply, under chloroform, in order to perform the necessary small operative procedures, and then allowing it to revert to a condition of light anæsthesia for purposes of the experiment. On the strength of these experiments it was concluded that the mammalian heart was made irritable by chloroform, that the irritability was raised under light anæsthesia and lowered under deep anæsthesia, and that a group of conditions (or exciting causes) which tend to stimulate the ventricles can make this irritability manifest.

It would appear, however, from the experiments to be now described that the irritability is not readily developed if the animal inhales only a low percentage of chloroform from the beginning, that is to say, if the heart has never been more than very lightly affected. This form of light chloroform anæsthesia may, for the purposes of this paper, be termed "primary" light anæsthesia.

Illustrative Experiment.

Cat. The preliminary operations were performed under the influence of ether, administered by artificial respiration. The vagi were cut in this experiment—rate of heart beat immediately before section 240 per minute and the same immediately after section.

10.41 a.m. A faint corneal reflex. Blood pressure 170 mm.; 0.4 per cent. chloroform vapour substituted for the ether vapour.

* A grant in aid of this research was made from the Graham Research Fund, University of London.

- 10.56 a.m. Active corneal reflex. Blood pressure reduced to 108 mm. and heart beat 210 per minute. 0.064 mgm. adrenalin injected into saphenous vein after 15 minutes inhalation of 0.4 per cent. chloroform. The blood pressure rises to 196 mm. and beat rate to 260 per minute, but the manometer curve shows a continued perfect regularity of the heart beat (Fig. 1). The percentage of chloroform was then gradually increased up to 2 per cent..
- 11.1 a.m. Chloroform 2 per cent.. No corneal reflex. Blood pressure 60 mm.; heart beat 156. Chloroform gradually reduced to 0.4 per cent..
- 11.11 a.m. 0.4 per cent.. Blood pressure 108; pulse rate 192.
- 11.16 a.m. 0.4 per cent.. Fairly active corneal reflex. 0.064 mgm. adrenalin injected. The heart beat assumed the typical character of a multiple tachycardia, terminating in ventricular fibrillation (Fig. 2).

This experiment was repeated under precisely similar conditions with a precisely similar result.

In a third experiment in which 0.5 per cent. chloroform was administered for 15 minutes, and in which the vagi were intact, extrasystoles followed on the first injection of 0.064 mgm. adrenalin, interspersed with regular beats during a period of 45 seconds, but for a few seconds only in a moderately high grade of complexity. The chloroform was then increased to 1.2 per cent., and subsequently reduced to 0.5 per cent., when the injection of the same quantity of adrenalin caused ventricular fibrillation.

On repetition of this last experiment a multiple tachycardia resulted on the first injection followed by momentary fibrillation. Permanent fibrillation resulted from the second injection.

In a fifth experiment (vagi intact), 0.3 per cent. chloroform was first administered for 31 minutes, sufficient ether being continued simultaneously to ensure immobilisation of the animal. Adrenalin was then injected and resulted in an acceleration of the heart beat from 108 to 216 per minute, with the advent of a few extrasystoles only. The chloroform was then increased up to 2 per cent. and again reduced to 0.3 per cent., ether being administered simultaneously as before. The second injection of adrenalin resulted in a multiple tachycardia and fibrillation with subsequent recovery.

Broadly speaking, it may be said that 0.5 per cent. chloroform given for 15 minutes and *never exceeded*, may or may not sensitise the ventricles sufficiently to react intensively to adrenalin. Lower percentages than this do not properly sensitise the ventricles, even if given for longer periods.

The foregoing experiments were suggested by a paper published by E. Nobel and C. J. Rothberger in 1914,⁴ detailing electrocardiographic observations on animals under chloroform. The authors state in their introduction that "our researches should not be regarded as a test of Levy's

statements as we, for reasons to be presently mentioned, frequently deviated from the methods chosen by him; on the same ground our partly differing results should not be accepted off-hand as a contradiction of Levy's assertions." Nevertheless the deductions drawn from these experiments are sufficiently at variance with the conclusions of Lewis and myself, that they cannot be passed over without comment. To quote the authors further, "Contrary to Levy we have almost entirely worked on tracheotomised animals, and thereby not only obviated the stimulation of the sensory nerves of the upper air passages by the chloroform, but likewise apparently the asphyxia which may be induced partly by the increased secretion of mucous, and partly by the mask covering the head." To this supposed

Fig. 1.

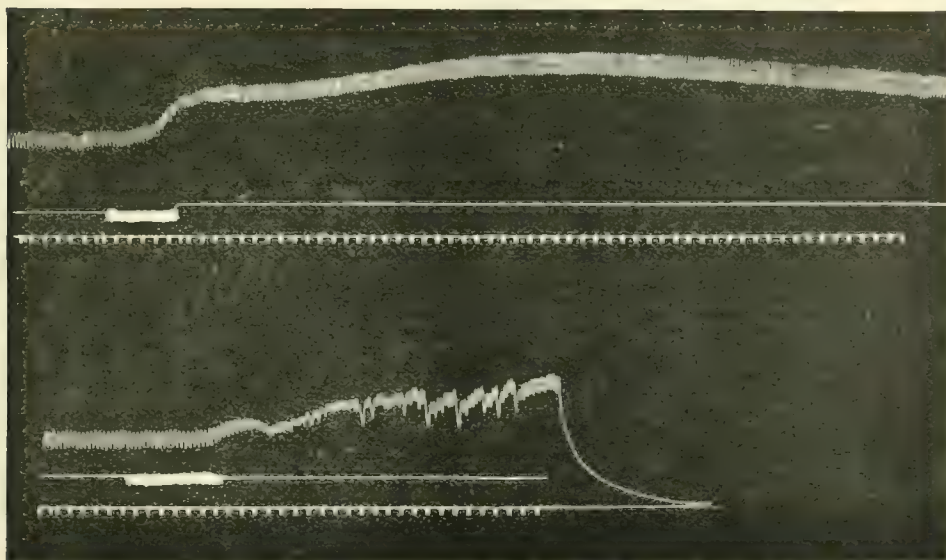


Fig. 2.

Figs. 1 and 2. Two Hürtle curves from the same cat, illustrating the effect of the intravenous injection of 0.064 mgm. of adrenalin (Fig. 1) under primary light chloroform anaesthesia; (Fig. 2) under secondary light chloroform anaesthesia. The signal marks denote the injection. In Fig. 1 the signal line has been adjusted to zero pressure; in Fig. 2, the time line. For full description see text. The time-marker is in seconds.

asphyxial element they attach considerable importance, but in this supposition the authors are entirely in error, for not only were my conditions always carefully regulated to avoid it (see page 322 of my article²), but likewise a considerable proportion of my experiments were performed under artificial respiration. The reaction shown in Fig. 2 was obtained under artificial respiration, as likewise was the entire series of five experiments described in this paper. In their series of experiments on cats Nobel and Rothberger first performed the necessary operative procedures under ether

narcosis; the animals were allowed to come round and then chloroform was administered in a low percentage. That this is an unsatisfactory method of studying ventricular phenomena under light chloroform narcosis has been shown by the experiments I have described above, but these observers fell into a yet more grave error in their technique. The form of inhaler they employed was the same as was used in my experiments; it was supplied at Dr. Nobel's request and the proper method of using it for animals described. Nobel and Rothberger, however, connected the trachea tube directly to the inhaler, allowing expirations to escape by a side tube under water. This apparatus was primarily constructed to work by the suction of human respiration, and the relatively feeble respirations of the cat are quite insufficient to set up a proper draught over the chloroform. I have performed an experiment with the apparatus used in this manner, and I found that after a fifteen-minutes administration of a nominal 0.5 per cent. chloroform vapour that the blood pressure had not dropped at all (*c.f.* experiment on page 105), and that the injection of adrenalin only set up a few irregularities in the tracing such as are frequently observed under ether narcosis alone. Further I found that even with a nominal 3 per cent. vapour it was impossible to maintain efficient narcosis. I conclude that with a setting of the index hand at 0.5 the animal only inhales the barest trace of chloroform vapour, if, indeed, any at all.

The whole of their series of experiments upon cats are adversely affected for this reason. It is impossible to decide the actual degree of chloroform anaesthesia under which the observations were made, and in certain cases there is little doubt the animals inhaled practically no vapour at all.

A further series of experiments was performed upon dogs. In these animals the respirations are more powerful and no doubt the vapour inhaled by natural respiration through a tracheal tube may have some approximation to that indicated on the index scale, and in fact the results obtained were more significant than in the case of the cats; complex tachycardias were obtained, and in one instance permanent ventricular fibrillation resulted. There are, however, two further sources of error in these experiments which invalidate the observations. The "light anaesthesia" is again that of primary light anaesthesia, which is a different condition to that under which my experiments were performed. Secondly, the dogs were morphinised, in order, as the authors state, to increase vagal activity. They were heavily morphinised, 0.25 gramme being injected subcutaneously, and apparently to a degree sufficient to allow of the preliminary operations being performed without further narcosis. Under such circumstances I regard these experiments as being of an entirely different order to my own, which were performed under the simplest possible conditions. The effect of increased vagal tone is to diminish the liability to ventricular irregularities, and distinctly affects the incidence of ventricular fibrillation adversely (see page 357 of my article², and page 325 of another article³). Whereas Nobel and Rothberger only obtained permanent ventricular fibrillation in a single

dog under these conditions, they found that by following up the adrenalin injection by 1.5 mgms. of atropin the ventricular tachycardia frequently terminated in fibrillation, thereby, I think, fairly demonstrating the error of their method, for the atropin may be regarded as neutralising the increased vagal tone induced by the morphia. In this connection it is noteworthy that atropin alone is capable of producing, in the lightly chloroformed animal, a multiple tachycardia generally terminating in fibrillation, as pointed out on page 317 of my paper,³ a fact of which the authors appear to be unaware.

In their "discussion" the authors make the following statements, which may certainly be taken to contradict my results:—

(1) "Whilst Levy always found arrhythmia to occur in normally breathing cats under light anæsthesia, we have always missed this in tracheotomised dogs and cats." This is not a correct statement of my conclusions. My statements in this regard are quite clear, and show that the onset of the arrhythmia is conditioned by the decrease or removal of the chloroform. "It is not my view that chloroform in a particular degree of concentration can initiate a specific form of irregularity, but the onset of irregularities is, I believe, conditioned by the *change of cardiac state* involved in the progress from deep to light anæsthesia." Again,² "the heart may be maintained beating at a perfectly regular rate even when lightly anæsthetised so long as the anæsthesia is a level and unchanging one, and no other disturbance is at work, such as may arise from sensory stimulations (see page 365)." The conditions of Nobel and Rothberger were certainly prejudicial to eliciting these irregularities, that is to say, of a primary light unchanging chloroform anæsthesia, of probably infinitesimal degree in cats, and complicated with morphine narcosis in dogs.

(2) "Stimulation of sensory nerves has further in Levy's researches led to arrhythmia under light narcosis, whereas in our researches on tracheotomised animals it was ineffective." Similar objections hold good in this instance. Nobel and Rothberger performed three experiments with negative results, whereas I have found reflex irregularities to occur with great constancy in over fifty observations on cats under secondary light chloroform anæsthesia. I have recorded positive results also in animals under artificial respiration, so that it is evident that the negative results were not conditioned by freedom of pulmonary ventilation, but solely by the adverse influences referred to.

(3) "The injection (of adrenalin) under light narcosis almost always led to a disturbance of rhythm; this was in our experiments altogether less intensive than in Levy's, who says that 0.064 mgm. adrenalin almost certainly led to ventricular fibrillation under light narcosis." The experiments described earlier in this paper demonstrate not only how this divergence may arise, but likewise that intensive results are obtained perfectly readily even when the lungs are freely ventilated. A statement is likewise made several times in the body of the paper that adrenalin caused

a higher degree of irregularity under deeper narcosis than under light ; this is no doubt partially correct under the conditions of primary light anæsthesia employed, but entirely contrary to what is observed following fully established anæsthesia. At the same time it is doubtful whether what Nobel and Rothberger describe as deep anæsthesia was really such, for they were guided by the readings of the index scale of the inhaler, which, as employed by them, could not work accurately.

The authors further do not appear to agree that chloroform makes the ventricles irritable in the sense employed by me, and they regard the adrenaline effect as due to a combined vagus and accelerator action; in fact, they are inclined to bring the chloroform phenomena into relation with an action upon the hypothetical tertiary "Reizbildungszentren" which are frequently referred to in Rothberger and Winterberg's writings. It is, however, certain that the adrenalin reaction occurs independently of all extraneous nervous action ; this point is definitely settled by experiments already recorded by me (see page 330 of my article²), but to which Nobel and Rothberger make no reference.

It is noteworthy that in certain experiments in which our experimental methods were identical, there is no divergence of opinion. Thus Nobel and Rothberger agree that adrenalin does not disturb the normal sequence of the heart beat in an animal not under an anæsthetic, and it is interesting to have an electrocardiographic confirmation of this fact. They also demonstrate the nature of the minor ventricular irregularities caused by adrenalin in the etherised animal, to which I have referred in my writings. However, to sum up, Nobel and Rothberger's experiments are generally vitiated by a method of primary light chloroform anæsthesia which fails to sensitise the ventricles, by a faulty method of administering percentage vapours, and by the preliminary use of morphia. Their peculiar results are due to these conditions, and not to the fact, as they presume, that their animals were tracheotomised.

REFERENCES.

¹ LEVY and LEWIS. *Heart*, 1911-12, III, 99.

² LEVY. *Heart*, 1912-13, IV, 319.

³ LEVY. *Heart*, 1913-14, V, 299.

⁴ NOBEL and ROTHBERGER. "Über die Wirkung von Adrenalin und Atropin bei leichter Chloroformnarkose." *Zeitschr. f. d. ges. exper. Med.*, 1914, III, 151.

ATTACKS OF PAROXYSMAL TACHYCARDIA FOLLOWING ATROPINE.

BY GIOVANNI GALLI.

(*Cardiac Department, Military Hospital of the Celio, Rome.*)

IN an article, which appeared in this Journal (Vol. I), Lewis has made a careful study with regard to the influence of belladonna upon the frequency of paroxysmal attacks. The drug was first administered (in the form of four-hourly doses of *Tinct. Belladonna m. x.*) on the evening of the 27th February, and was continued until the evening of the 4th of March, when the dose was increased to *m. xx* up to the night of the 7th of March. The proportions of counts of fast and slow rate before and after the administration of the drug, were practically identical. Lewis concluded, therefore, that the vagus has no influence in the production of the paroxysms, and that the view that the attacks may be due in certain instances (as explained by Bouveret, Notnagel, and others) to a withdrawal of vagal influence receives no support. My own evidence, such as it is, definitely points in a direction contrary to such a conclusion.

In opposition to the opinion of Lewis I am in a position to cite a case, in which it was possible to provoke artificially typical attacks of paroxysmal tachycardia.

A soldier, whose civil trade was that of a farmer, complained of having suffered since the age of 12 from tachycardia appearing and disappearing in an unexpected manner. These attacks lasted a few minutes, at the maximum a quarter of an hour, and they were provoked by emotion and other causes unknown to the patient. For two years this soldier had a slight affection of his left ear, which discharged pus. The otological examination proved the complete destruction of the ear tampon, but failed to show the cause and nature of the disease. This man having been declared fit for service, was at the age of 20 enlisted in the cavalry, but after a few days was found to be unfit for service, owing to cardiac trouble. At 21 years of age, having again been proclaimed fit for military service, he was enlisted in the 56th infantry regiment at Belluno, but after a month of service obtained twelve months' sick leave: on expiration of this he was finally discharged. At 24 years of age (*i.e.*, July, 1916) he was submitted to a new medical board and declared once again fit for service, but, after a few days,

owing to tachycardial trouble, he obtained a further two months' sick leave. On his return to his unit he again reported sick and obtained a further two months' extension of leave. In February, 1917, he was judged permanently unfit for the firing line, and was sent to the war zone to be incorporated in a regiment in charge of prisoners of war. On his arrival there he was submitted to another medical visit and sent to Hospital No. 047, for observation. Within a few days he again reported sick, and after having been to various hospitals, and having obtained various extensions, and also having been submitted to various medical boards, he finally arrived at my department. The service given by this soldier was but little. The fact that the examination of the heart was negative made his medical officer suspect that he had to deal with a malingerer, with a very little will for any work, a suspicion enhanced by the presence of an ear-lesion and by the impossibility of confirming the tachycardial attacks of which the patient complained during the night and also at daytimes for a few minutes.

Having applied various expedients to provoke the tachycardia (for example, the Valsalva experiment), which, however, never succeeded, I also submitted him to muscular efforts. I tried to personally impress him, by showing to him, with his consent, other wounded under treatment, but without being able to provoke in him the tachycardial effects, and in my thoughts I had already judged the patient to be a malingerer. However, one morning, during the medical round, I personally observed the beginning of a spontaneous attack. While I was approaching the bed of the patient, he signalled to me that he was at that moment being subjected to an attack. The beating of his pulse was in reality at about 200 per minute, and while I was making the necessary preparations for graphic record the crisis ceased. A similar episode happened a second time: I, therefore, thought of availing myself of atropine. On the 19th February I injected the patient hypodermically with atropine (1.0 milligram) at 9.45. The beating of the pulse was at 90 previous to the injection and the breathing was 20 per minute. Four minutes after the injection had been administered the beating of the pulse and breath started to decrease. Such decrease (to about 73) lasted for about 15 minutes, after which the average frequency increased to its previous level. Suddenly, 43 minutes after the injection, the pulse beat increased to 215, with an increase also of the respiration (to about 30). This suddenly dropped to 110, and within three-quarters of an hour decreased to 75. The decrease was more accentuated at night time, and at 10 p.m. the pulse rate was 61.

It is to be observed that the pulse rate decreased after the injection, and this I have noted in other cases, when I have administered atropine. In using atropine for intravenous injections the preliminary decrease in my experience does not occur. By administering atropine to the patient by the mouth it was possible also to obtain a typical attack of paroxysmal tachycardia, save that the attack came later (say one hour after having taken atropine). At 8.43 o'clock, on the 9th June, 20 drops of atropine

were given by the mouth (*i.e.*, 1 milligram of atropine). After this the pulse declined as far as 64, and then suddenly increased to 190, 57 minutes after the administering of atropine. The attack lasted for a very short time, *i.e.*, less than two minutes, and then it ceased instantaneously, the frequency falling to 70. The attack had scarcely ceased, when about one minute afterwards a second one started (Fig. 1), also typical, the onset being sudden

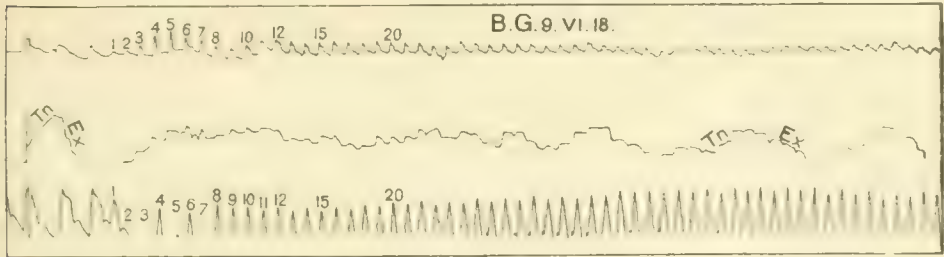


Fig. 1.

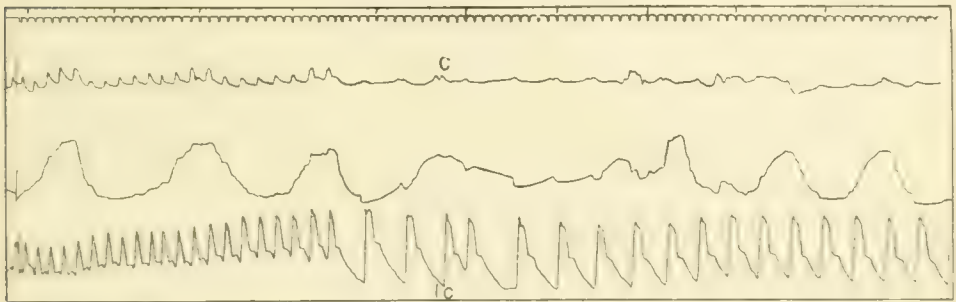


Fig. 2.

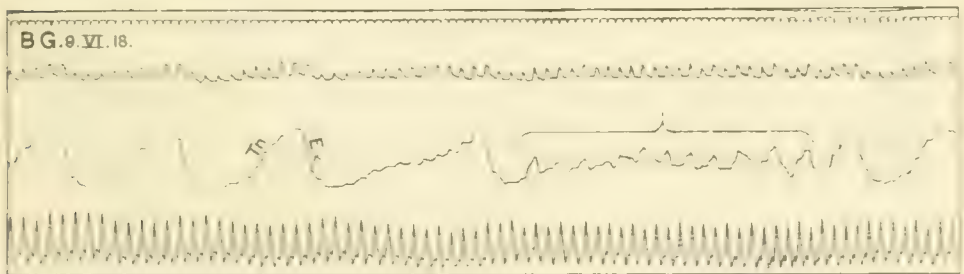


Fig. 3.*

and the frequency 205 beats per minute. After this, the pulse oscillated, always maintaining itself high. At 10.5 a tendency was noted for the rate to decline. The pulse having gone down to 147, and immediately afterwards it accelerated again to 195, when, however (at 10.17), it fell suddenly to 70 (Fig. 2).

* These three curves have been redrawn.

A curious increase in the rate of respirations during the attack is shown in Fig. 3. The breathing is frequent, superficial and irregular. I have had occasion to observe this breathing on many occasions, also in other patients. The group shown in Fig. 3 demonstrates rapid movements having a "crescendo" quality. I have already described in a man 100 years old a type of respiration similar to the above-mentioned case. (*Archives des maladies du cœur*, 1919, Février.)

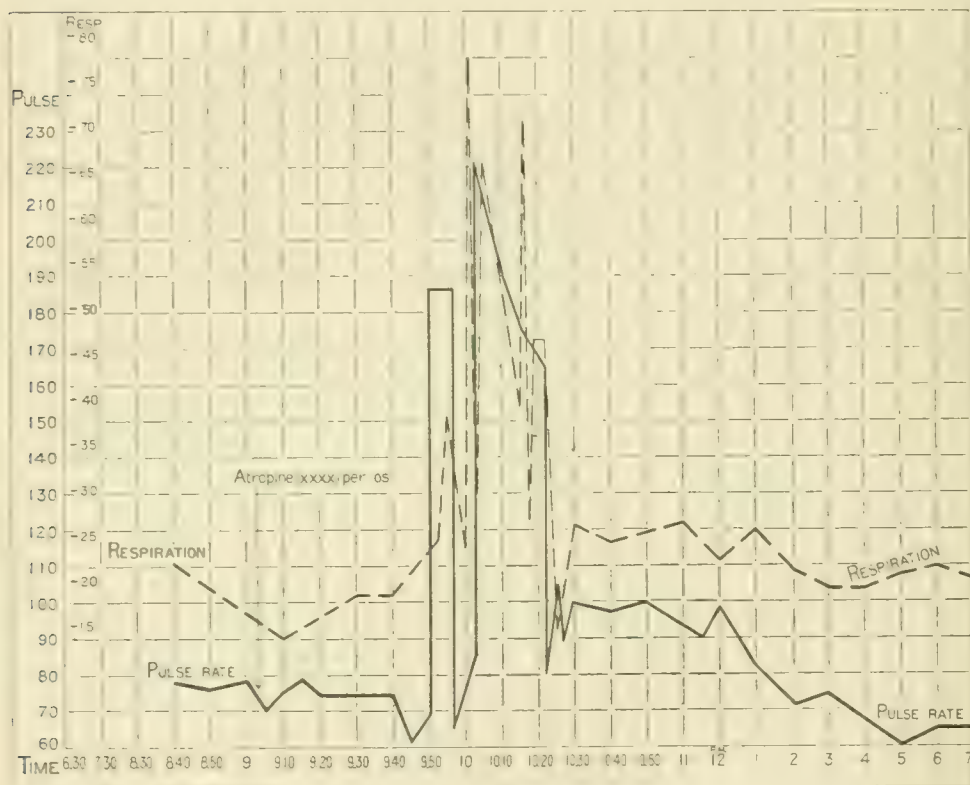


Fig. 4.

Fig. 4 is a diagram of an attack which happened on the 22nd June, 1918, and followed 40 drops of atropine (2 milligrams). Here also the attack is a double one, but different in its detail from that of the 9th June. The crisis begins (about 45 minutes, instead of 57 minutes after atropine) and lasts five minutes less. It is less oscillating, and the rate of breathing follows generally that of the pulse. Fig. 5 shows the progress of an attack, caused by 30 drops of atropine. This crisis has an aspect of more stability and is of a long duration (2½ hours). The start and the end of the paroxysm



Fig. 5.

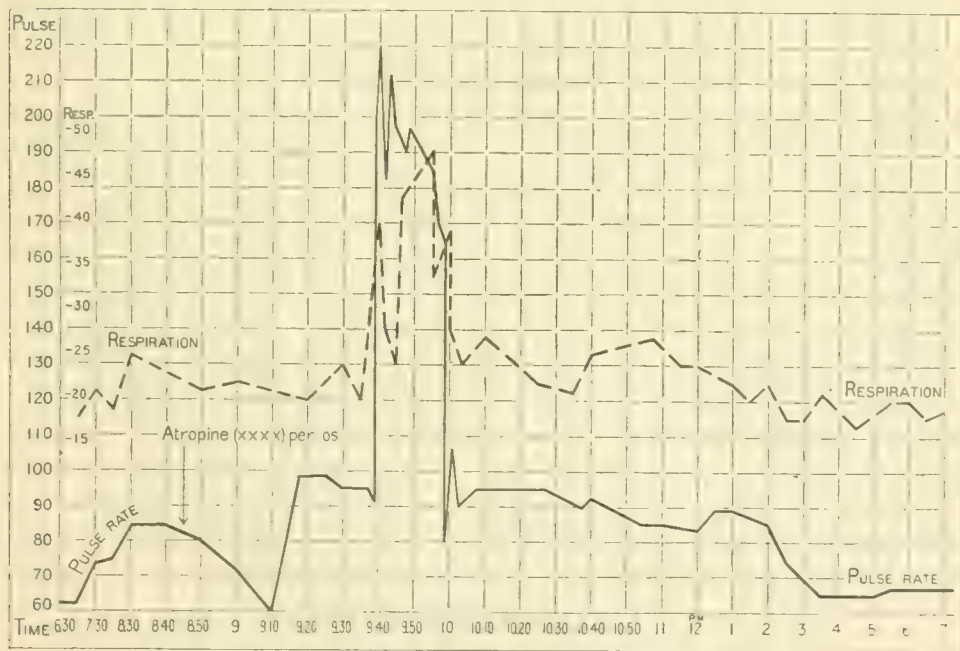


Fig. 6.

are abrupt. The breathing follows the pulse, but the frequency of the breathing is less high than in Fig. 4, where it is more than doubled. The attack represented by Fig. 6 was caused by 40 drops of atropine, but this time the attack lasted 12 minutes less. An attack, provoked on the 2nd of June with 20 drops of atropine, started about one hour after the administering of the drug. It was double, as was that illustrated in Fig. 4, but of shorter duration, lasting only 12 minutes. The appearance and duration of these attacks was evidently controlled by the quantity and rapidity of action of the atropine. The accumulation of atropine in the blood is more favoured, according to the gastric potentiality (which varies from person to person and also in the same person), and to the elimination of the kidney, which also varies.

When the atropine is administered in massive doses the tachycardial effects are more evident and constant.

To conclude, therefore, I am in a position to affirm that tachycardial attacks can be provoked in man by atropine, and that Lewis has failed to obtain these attacks either because he administered the drug continuously, or owing to the insufficient quantity administered. It is to be observed that Lewis's patient failed to show a reaction of the heart to atropine of the usual or physiological type.

AN ELECTROCARDIOGRAPHIC PLATE STUDIED BY MEANS OF THE COMPARATOR.*

BY THOMAS LEWIS.

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THE purpose of this article is twofold. In the first instance it intends to illustrate a method of finely measuring electrocardiographic curves which has been in routine use in my laboratory for some years. In the second place it intends to explore certain factors of error which occur in measuring plates taken by means of the galvanometric outfit† now in general use in this country.

The photographic plate chosen for the purpose of illustration was taken some years ago from a patient who was the subject of auricular flutter, the auricles beating at 268 and the ventricles at 134 per minute. This plate forms one of a series of curves measured and reported in the succeeding article, which deals with the regularity of the heart's movements in auricular flutter.

The curve has been measured by means of the Lucas comparator, a description of which will be found in the *Journal of Physiology*, Vol. XXXIX, page 217. The comparator consists essentially of two small microscopes (2.3 inch objectives) set parallel but some inches apart on a single stand. This stand with its two microscopes slides nicely from side to side upon a rigid bar beneath which are, on the one side, a carrier which holds the plate to be measured and, on the other side, a glass scale 15 centimetres long, each centimetre being subdivided into 1,000 parts.[‡] The cross wires of the left hand microscope are adjusted to lie over a point on the photographic plate from which a reading is desired, and the reading is obtained at the point where the cross wires lie on the scale as viewed through the right hand microscope. To measure a distance between two points on the photographic plate, readings are taken from each and the subtraction gives the number of scale divisions corresponding to the interval. This number of scale divisions is subsequently converted into seconds by measuring the number of scale divisions lying between time marks.

* Undertaken on behalf of the Medical Research Council.

† The outfit supplied by the Cambridge Scientific Instrument Company.

‡ Actually subdivided into 100 parts, the thousandths are judged.

TABLE I.
Tabulated measurements of a curve.

Time lines.	Time lines apart in scale division.	R.	Time lines to R.		R-R in seconds.
			Scale divisions	Seconds.	
500					
	518*	551	51*	-0197+	
1018			467*	-1803+	
1542	524			-2	.4428
	522	1705	163	-0625	
2064			359	-1375	
2584	520			-2	.4469
	521	2869	285	-1094	
3105			236	-0906	
3612	507			-2	.4469
	494	3998	386	-1563	
4106			108	-0437	
4598	492			-2	.4529
5087	489			-2	
	500	5110	23	-0092	
5587			477	-1908	
6097	510			-2	.4520
	513	6254	157	-0612	
6619			356	-1388	
7132	522			-2	.4573
	525	7443	311	-1185	
7657			214	-0815	
8173	516			-2	.4468
	502	8588	415	-1653	
8675			87	-0347	
9169	494			-2	.4472
9663	494			-2	
	497	9694	31	-0125	
10160			466	-1875	
10654	494			-2	.4438
	497	10794	140	-0563	
11151			357	-1437	
11654	503			-2	.4472
	514	11920	266	-1035	
12168			248	-0965	
12684	516			-2	.4519
	516	13085	401	-1554	
13200			115	-0446	

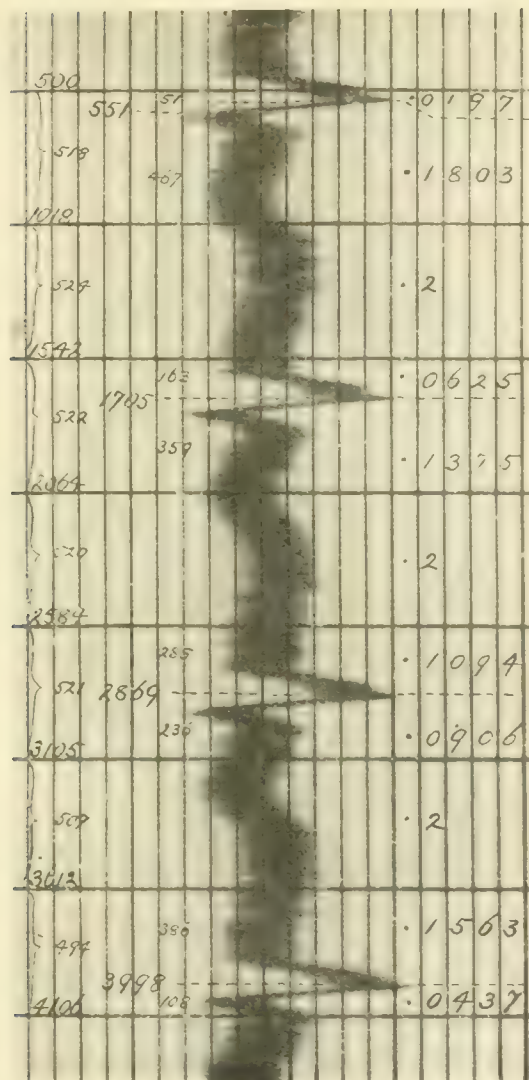


Fig. 1. Portion of the measured curve. The figure is an enlargement and shows the first three R - R cycles of the table. Below the curve (to the left as it is published) the scale readings of R_s and time lines are given (larger figures) and also the distances between time marks, and each R and its adjacent time marks, in scale divisions (smaller figures). Above the curve the time distances of R to adjacent time marks are given in seconds. The numbers are arranged in the same fashion as in the table on the opposite page.

The actual procedure adopted varies somewhat according to the information required, but is sufficiently illustrated by a single example. From the plate, it is desired to ascertain the time distances between adjacent summits *R* as accurately as possible. The plate is inserted into the comparator and the microscopes are focussed and adjusted so that the first time line coincides with a convenient centimetre mark on the scale; in this instance the reading for this time marker is 500. This reading is noted in the first column of a tabular statement (see Table I) and, as the microscope is moved across the plate, successive readings are taken from all the time marks and summits *R* as they appear. The readings of the time lines (500, 1018, 1542, 2064, etc.) are entered in the first column, the readings of *R* (551, 1075, etc.) are noted in the third column. In tabulating the readings, the level at which they are placed in the table is governed by their values, irrespective of the column in which they lie. Finally, the microscopes are returned until the first time line lies beneath the cross wires and the reading 500 is checked to ascertain that during the process of measurement the plate still occupies its original position relative to the scale.

From these readings the necessary information is obtained. Our routine is as follows:—The number of scale divisions between adjacent time lines is calculated by subtraction; it is necessary only to calculate those between which a reading for the summit *R* falls (heavy figures in the second column of the table). In the fourth column the number of scale divisions lying between *R* and each of the two time lines between which it falls is entered. The method and object of this tabulation is illustrated in Fig. 1, which is an enlarged print from the first three cycles of the plate. The numbers in the fourth column are now converted into seconds by multiplying by 0.2 and subsequently dividing by the corresponding figure lying in the second column.

Thus :—51 scale divisions in the fourth column = $\frac{51 \times 2}{518 \times 10} = 0.0197$.

These readings in seconds are inserted in the fifth column. In practice the number in the fourth column is multiplied by 2, and the division is made by slide rule.

The subtractions of the second and fourth columns are checked as they are made, the pair of numbers (marked by asterisks) in the fourth column being together equal to the corresponding single number in the first column. The slide rule calculations are checked by noting that the figure in seconds, when added to that to which it stands next, sums to 0.2000 of a second (see †† in table). In the same column the figure 0.2 is inserted at appropriate points, where a complete one-fifth second interval is included between two *R* summits. The *R*-*R* distances in seconds are now obtained by adding together the appropriate figures in the fifth column.

The margins of error.

Error arising in judging the time position of R. In curves such as this, in which *R* is sharply pointed at its summit, readings of its position relative to the measuring scale are not subject to much error. The cross wires are placed at the approximate centre of the summit, and the scale shows a reading of 551: by moving the position of the cross wires and taking fresh readings it is seen that the reading 549 or 553 would not correctly represent the centre, and that the time reading lies somewhat between 550 and 552. The greatest possible error if due care is exercised amounts to one of the smallest scale divisions in either direction. Actually it is less in the average, as is shown by repeated readings of all the *R* summits of the plate. Thus, in two successive observations the readings were identical in five instances, varied one scale division in six instances and two scale divisions in one instance. For security we take the maximal error of one scale division in either direction. Where one-fifth second measures 500 scale divisions, the maximal time error introduced is $\frac{1}{500} \times 1.5$ in either direction, or \pm or -0.0004 of a second. Supposing that this maximal error occurred for two adjacent *R* summits, and that in the two instances it was opposite in direction, which is unlikely, then the total error for the *R-R* time distance would be 0.0008 of a second in either direction.

Error arising in judging the true position of the time lines. The error in judging the position of the time lines is less than for *R*. In a repeated reading of the plate, the readings were identical in the case of 15 time marks and varied by one scale division in 11 instances. Allow a maximal error of 1 scale division, and the error so introduced in measuring a portion of the *R-R* time interval will vary according to the magnitude of the latter within certain limits. Thus, if the difference between adjacent time lines measures 500 on the scale and the magnitude of the interval to be measured is 23 scale divisions, the error will be minute, namely, $\frac{23}{500 \div 5} - \frac{23}{501 \div 5} = 0.00920 - 0.00918 = 0.00002$ of a second. As the magnitude of the interval measured rises to 500 scale divisions (or one-fifth second) the error will rise to $\frac{500}{500 \div 5} - \frac{500}{501 \div 5} = 0.2000 - 0.1996 = 0.0004$ of a second, or 0.0002 of a second in either direction. If the same maximal error were to occur for two time lines* and were opposite in direction in the two instances, the total possible error from this source would be 0.0004 second in either direction.

* Actually it could not, because the two intervals measured in computing an *R-R* distance never amount in this curve to 0.4 second, the highest amount being 0.2573 of a second (6th cycle).

Error in the time marker. The time marker used is a rotary time marker controlled by a standard double-pronged tuning fork of 50 vibrations per second. The error which concerns us is the actual variation in the time distance between adjacent time lines. These time lines purport to lie at a time distance of 0.2000 of a second from each other.

The error due to variation is ascertained by recording these fifth-second time lines side by side with the movement of the tuning fork* on rapidly travelling plates and by taking the vibrations of the tuning fork as the standard. Twenty-five fifth-second intervals have been measured in this fashion and the average error was found to be 0.0006 of a second. On one occasion the supposed fifth of a second measured 0.1985 of a second, on one occasion 0.2015 of a second. There is an occasional maximal error of 0.0015 of a second in either direction in measuring a distance of 0.2 of a second. The fault is due to lack of precise harmony between the tuning fork and the rotary wheel. Now a continuation of such an error in several successive rotations of the wheel would destroy the unison between tuning fork and rotary marker and would bring the latter to a standstill; consequently such errors do not occur. The error in estimating time lines adjacent to that in which a large error occurs is either a relatively negligible quantity or is reversed in direction. Although the error arising in measuring 0.2 of a second may be as great as + or - 0.0015 of a second, the error in measuring 0.45 of a second (R - R cycles) is not two and a half times as great, for the error in measuring three successive time lines does not in any case exceed 0.0017 of a second. We may therefore regard + or - 0.0017 second as the maximal error introduced from this source.

Error arising from inequalities in the speed at which the plate travels. Gross error of this kind is obviated by frequent reference to the actual time lines. Minor error arises in the following fashion. In using the measured distance between adjacent time lines for the purpose of calculating the R - R distance it is assumed that the movement of the plate is quite uniform in the space between one time line and the next. Now this is clearly not actually the case, and an error arises.

To illustrate, let us take an extreme instance of the present curve, namely, the length of the 3rd R - R cycle measured. The R - R distance subtends a whole one-fifth second interval and a portion of each adjacent one-fifth second interval. The three intervals between the time lines concerned measure 521, 507 and 494 scale divisions respectively; the speed at which the plate travels is slowing. The numbers actually used in calculating R - R in seconds, are 521 and 494, the figures in seconds being 0.0906, 0.2 and 0.1563. In this calculation it is assumed that the plate travel

* The error introduced by variations in the tuning fork itself is negligible for our purpose.

is uniform through each one-fifth second concerned, and that the change of rate occurs abruptly as each one-fifth second is marked. The slowing is clearly gradual throughout the whole period. If the slowing began in the

TABLE II.

Values taken.	One extreme.	Other extreme.	Error cannot exceed and is certainly less than.
4428	4404	4428	$\begin{cases} +0000 \\ -0024 \end{cases}$
4439	4469	4477	$\begin{cases} +0008 \\ -0000 \end{cases}$
4469	4429	4494	$\begin{cases} +0025 \\ -0040 \end{cases}$
4529	4529	4533	$\begin{cases} +0004 \\ -0000 \end{cases}$
4520	4482	4524	$\begin{cases} +0004 \\ -0008 \end{cases}$
4573	4547	4580	$\begin{cases} +0007 \\ -0026 \end{cases}$
4498	4424	4482	$\begin{cases} +0014 \\ -0044 \end{cases}$
4472	4472	4478	$\begin{cases} +0000 \\ -0006 \end{cases}$
4438	4438	4454	$\begin{cases} +0000 \\ -0000 \end{cases}$
4472	4454	4495	$\begin{cases} +0023 \\ -0018 \end{cases}$
4516	4515	4519	$\begin{cases} +0004 \\ -0000 \end{cases}$

one-fifth second which measures 521, and that is possible, the rate of travel during the latter part of this one-fifth second interval would be represented by a figure intermediate between 521 and 597. We actually take the measurement 521 for purposes of calculation and obtain for the corresponding

part of the R - R time length the value $\frac{236}{521 \times 5}$ or 0.0906 of a second. If we took the measurement at the other extreme, namely, 507, we should obtain the time value $\frac{236}{507 \times 5}$ or 0.0931 of a second. Somewhere between these two extremes the true value lies. It almost certainly lies nearer to 0.0906 than to 0.0931 of a second. It should be understood that the

TABLE III.

Maximum errors from different sources and gross maximum error.

R - R cycles.	From travel of plate.	In judging R .	In judging time lines.	From time- marker.	Gross maximal.	Actual values taken.	Possible values.
1	-0.0000 +0.0024	or	or	or	+0.0029 -0.0053	4428	4457
2	+0.0008 -0.0000				+0.0037 -0.0029	4469	4472
3	+0.0025 -0.0040				-0.0054 -0.0069	4469	4472
4	-0.0004 +0.0000				+0.0033 -0.0029	4529	4500
5	+0.0004 -0.0038				-0.0033 -0.0067	4520	4472
6	+0.0007 -0.0026				+0.0036 -0.0055	4573	4518
7	+0.0014 -0.0044				+0.0043 -0.0078	4468	4472
8	+0.0006 -0.0000				+0.0035 -0.0029	4472	4472
9	-0.0016 +0.0000				+0.0045 -0.0029	4438	4472
10	+0.0023 -0.0018				-0.0052 -0.0037	4472	4472
11	-0.0000 +0.0004				+0.0029 -0.0033	4519	4484

divergence between 0.0906 and 0.0931 of a second expresses the very highest possible error, namely + 0.0025 of a second, and that the actual error is certainly much smaller than this, though we cannot say how much smaller.

A similar error arises in calculating the last phase of the same R - R time interval, for we use the measurement 494 in our calculation, whereas the rate of plate travel is represented actually by a figure lying between 494 and 507.

The measurement 494, gives us a calculation time interval $\frac{386}{494 \times 5}$ or 0.1563 of a second; going to the other extreme and using the measurement 507 we

should obtain a time interval of $\frac{386}{507 \pm 0.5}$ or 0.7623 of a second. This divergence namely ± 0.0040 second, represents again the highest possible error, and is certainly too great, but by precisely how much we are unable to say.

In calculating the R - R period in seconds we have the following extreme alternatives, between which the true value lies :—

0.0906	0.0931
0.2	0.2
0.1523	0.1563
1 = 0.4429 of a second,	and 2 = 0.4494 of a second.

The value actually taken is 0.4469, and the maximal error is ± 0.0025 or ± 0.0040 of a second.

The example considered is the least favourable of the whole curve: the highest possible error from inequalities in plate travel for the remaining R - R cycles is less, usually much less. Using alternative and adjacent values for the one-fifth second time intervals throughout we arrive at the alternative values of the R - R cycles shown in Table II.

Possible errors from all sources.

We may now combine the maximal possible errors from all sources. They are stated in the accompanying table (Table III), in the last column but two. In this table I have given the accepted time values of the R - R intervals. In the last column, possible actual values are given: these are obtained by utilising gross maximal errors as corrections. Using these corrections to the greatest advantage the possible values of R - R cannot be rendered completely uniform in this column: it is certain therefore that the R - R time distances are variable. But the extent of the greatest variation cannot be stated precisely: it cannot be less than 0.0028 of a second: it cannot be greater than 0.0234 of a second. It certainly lies well toward the first figure, though the actual figure cannot be computed by the method used with greater precision than is here stated. We are able to ascertain the maximal possible errors for our curve, but from the data already given the probable errors cannot be given, beyond stating that they lie well below the maximal possible errors. To arrive at a more precise conception of the probable error in measuring this plate another method is adopted. It is to record upon a plate travelling at approximately the same speed and under other similar conditions, the vibrations of a 50 tuning fork, and to mark each 22nd vibration in the record. These marked vibrations, lying as they actually do 0.4400 of a second apart* are measured against the fifth second time lines. The divergence from 0.4400 of a second represents

* The error in the tuning fork vibration is negligible for our purpose.

the error in measurement. Three plates have been measured in this fashion, and the figures obtained were :—

Plate I. +4402, +4391, +4411, +4398, +4403, **+4386**, +4403, +4401, +4399, +4409, +4408, +4400.

Plate II. +4404, +4407, +4405, +4395, +4399, +4389, +4406, +4417, +4397, +4399, +4394, +4398.

Plate III. +4407, +4412, +4392, +4395, +4409, +4391, +4415, +4404, +4390, +4398, **+4418**, +4398.

The average divergence from 0.4400 is 0.0006 of a second ; the greatest divergence is +0.0018 and -0.0014 of a second. A divergence of 0.001 second or more is present in 8 readings out of 36, or 1 in 4 or 5. Now the factors of error in measuring these plates are the same as in reading the *R-R* distances in our patient : we may say, therefore, that the errors as expressed by the above figures represent the probable error in the values of the *R-R* distances accepted for our patient's curve. Comparing these figures of probable error with those of the calculated maximal error (Table III) we see at once that the probable error is very much less considerable than the maximal possible error. That is so, because the error arising from misjudging the position of *R* or time marker is usually almost negligible ; because the error arising from unequal speed of plate travelling on the one hand and from inequalities of the time marker on the other, usually fail to approach the maximal possible error at all closely ; and, lastly, because in calculating the gross maximal error all possible errors are combined in one direction, whereas actually they will in the great majority of instances fall in opposite directions and tend to neutralise each other.

The values for the *R-R* time-distances in our patient's curve may be accepted up to the third decimal point, it being understood that there is a probable error approximating to 0.0010 or 0.0015 of a second in one or other direction in the case of 1 cycle in 4 or 5. The fourth decimal point has little or no value except in so far as it shows the inclination of the third decimal point.

OBSERVATIONS UPON FLUTTER AND FIBRILLATION.

PART I.—THE REGULARITY OF CLINICAL AURICULAR FLUTTER.*

By THOMAS LEWIS.

(University College Hospital Medical School.)

CLINICAL auricular flutter, first described in patients by Hertz and Goodhart,¹ and so named by Jolly and Ritchie,² is a condition in which the movements of the auricle are extremely rapid, the actual rate of auricular contraction being in most cases in the neighbourhood of 300 per minute. As is now well known, the ventricle rarely keeps the auricular pace—it may do so occasionally—but responds by a half or quarter rhythm.

Now a similar condition to that found in the patient is seen from time to time in animal experiment;³ it may be provoked on occasion by faradic or rapid interrupted stimulation of the auricle, it may appear when the blood is contaminated by the injections of such poisons as glyoxylic acid; sometimes it appears spontaneously, by which it is meant that the disorder arises from causes unknown to us. As I have seen and recorded it in these several circumstances, the movement of the auricle is regular to a very exact degree.† The rapidity and regularity of the auricular movements as they are recorded in patients constitute the chief features of the clinical condition. When we observe a similarly rapid and equally regular movement of the auricle in experiment we are justified in regarding the experimental condition as of the same or similar nature as the clinical. The object of the experimental work is to throw light upon the human malady. But we are scarcely wise in applying the term flutter to a disorder of the heart produced experimentally unless the disorder in question shows similar characteristics to those which we observe in our patients. If it is stated, on the basis of the unaided senses that the auricle was seen to pass into flutter under certain conditions, it is hardly safe for us to regard this condition as necessarily analogous to those which may underlie clinical flutter. We need first to be certain that we are dealing in the experiment with a condition really identical with or akin to clinical flutter. But the term "flutter" has been applied to disorders encountered in experiment in which the rate of the auricular beating is very

* Observations undertaken on behalf of the Medical Research Council

† To be described in a subsequent communication.

high and in which there is an approach to regularity of movement.^{4 5} The degree of regularity to which I refer is one in which there is no very noteworthy degree of *irregularity* when quite coarse measurements are undertaken in slow travelling records or when the auricle is simply inspected. The purpose of the present short article is to draw attention to and emphasise the high degree of *regularity* in clinical flutter, and to suggest that the term "flutter" should be confined both clinically and experimentally to disorders in which this high degree of regularity obtains. There is no inherent objection in the employment of the term in experimental disorders in which the auricular movements may be or demonstrably are of a less orderly nature; but if by so employing the term in experiment we tend to identify these relatively irregular and rapid movements of the auricle with the relatively regular and rapid movements of the patient's auricle—and this tendency is, I think, in the circumstances inevitable—then our terminology may easily lead us astray.

Judging from considerable experience of clinical curves, measured without magnification, that the regularity of the auricular movements is very precise, I have thought it worth while to measure in a more exact fashion a number of unselected clinical curves and to place the results on record, so that the degree in which absolute regularity is approached may be recognised more universally.

I have chosen from my collection electrocardiograms from seven separate subjects, and in four of these cases have chosen curves which were obtained on different dates, and have submitted them to measurement by the comparator. The measurement of intervals between adjacent auricular systoles has not been practicable for my purpose, because the deflections which represent auricular systoles in these curves are not sufficiently acute to afford sharp measurement. It has been found more profitable to measure the intervals between ventricular systoles, using only such records as show a regular response of ventricle to auricle. The results are given in the accompanying table. The error of measurement has been sufficiently discussed in the preceding article; it may be taken that these measurements are correct to three places of decimals with few exceptions, and that exceptionally there may be an error of 0.001 or 0.0015 of a second in either direction.

The average variation of the V_s - V_s intervals ranges in different patients from 0.0009 to 0.0077 of a second. The average variation is usually a matter 1, 2 or at the most 3 thousandths of a second; in no case is it as much as 1 hundredth of a second. The maximal variation is usually less than a hundredth of a second; in some instances it somewhat exceeds a hundredth of a second; in a single instance it has amounted to a fiftieth of a second and a little more. These measurements are completely in accord with my experience of measurements without magnification. They mean that such variations as occur are beyond our powers of observation in unmagnified

curves taken at such rates that the ventricular beats are separated by distances of a half-inch or less, and that in most instances they are indistinguishable in records where the ventricular beats lie one inch apart from each other. The degree in which absolute regularity is approached is a very high one; but absolute regularity of the auricle is almost certainly approached even more closely than is represented by these figures, for we are measuring the *Vs-Vs* intervals, and not the *As-As* intervals. The method includes, therefore, not only variations in the *As-As* intervals, but any variation in the *As-Vs* conduction intervals which may be added to the first. There is a further consideration; our measurements, with the exception of three plates, indicate the spacing, not of successive auricular beats, but of alternate auricular beats; for in the case of all but three plates the response of the ventricle was to alternate auricular systoles. Although this method of measurement may not exaggerate our estimate of the average variation in the auricular cycles, it will certainly exaggerate our estimate of the maximal variation between individual cycles from time to time. For, if in a curve presenting 2:1 block, two relatively short intra-auricular cycles stand adjacent, and two relatively long intra-auricular cycles stand adjacent, the maximal variation for intraventricular cycles will be almost double that for interauricular cycles.

Conclusions.

Variations in the lengths of intra-auricular cycles in clinical auricular flutter average less than 0.0009 to 0.0077 of a second in curves which include 14 to 32 auricular cycles; they always average less, probably considerably less, than 0.01 of a second. The maximal variation of the cycles in the same curves is usually less, probably considerably less than 0.01 of a second; probably it may amount to, but does not materially exceed, 0.02 of a second on occasion.

It seems advisable that the term auricular flutter should be confined to disorders, be they clinical or experimental, in which comparable variations in the lengths of auricular cycles are found not to be exceeded.

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A POLYMYOGRAPH AND A COMPARISON OF THE CONTRACTION AND EXCITATION WAVES IN THE MAMMALIAN AURICLE.

By THOMAS LEWIS.*

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WITH THE COLLABORATION OF

H. S. FEIL, of Cleveland, and W. D. STROUD, of Philadelphia.

It has been proved in recent years that the excitation wave, which is associated with the normal contraction of the heart muscle, has its origin in the upper reaches of the sulcus terminalis in mammals and that it spreads from the immediate region of the sino-auricular node to the rest of the auricular tissue. The course which this wave of electrical disturbance takes in its passage through the auricular tissue has been the subject of detailed study, an account of which will be found in the full paper published from this laboratory by Lewis, Meakins and White in 1914.² These observations have shown that the wave spreads from its original starting point in a radial fashion, pursuing its course in an approximately regular fashion along the chief muscle bands which meet near the cephalic end of the sulcus. Thus, starting from this central point, the wave travels down the straight band of muscle which is called the tenia terminalis to reach the inferior vena cava, it travels to the left along the thick band of muscle which unites the ventral surfaces of the two auricles (intra-auricular band), it travels from the base to the apex of the right auricular appendix, and it travels up the superior vena cava against the blood stream.

This *excitation wave*, constituted as it is by an electrical disturbance, which is still imperfectly understood, but which is very readily recorded by means of sensitive galvanometers, is customarily regarded as the pioneer of the *contraction wave* which succeeds it. It is well known from experiment on voluntary muscle that an appreciable interval elapses between the appearance of the excitatory and the contraction processes. Therefore, it has been assumed that there is an interval between the same processes in heart muscle, an assumption which has been strengthened by the very obvious delay between the beginning of the electrocardiogram of auricle or ventricle and the corresponding contraction curves in the cold-blooded

* Working on behalf of the Medical Research Council.

heart and by the less obvious, though quite evident, delay of a corresponding kind, in the mammalian heart. But, so far as I am aware, there has been no attempt to obtain accurate measures of the actual interval between the two processes. An electrocardiogram taken at the usual sensitivity from the body wall does not signal the first appearance of an electrical disturbance in the heart. An electrode placed directly on a heart chamber does not necessarily signal the first appearance of the excitation process in that chamber; neither does a lever attached to the muscle of a chamber necessarily signal the beginning of contraction in that chamber: for both excitation and contraction waves travel, and take time to travel. Accurately to measure the interval between excitation and contraction processes, it is necessary to estimate exactly the beginning of these two processes at a given point of the muscle investigated.

It has also been assumed, on account of the constant association of the two processes during the systole of auricle or ventricle, that the course taken in travelling by the one is the course taken by the other.

The primary purpose of the present research has been to test the validity of the assumptions which I have discussed and, in the event of their proving legitimate, to base the conclusions upon the surer foundation of direct observation and accurate measurement.

The method which has been used to study the course of the excitation wave is one of very great delicacy: it is that of leading off by means of small non-polarisable electrodes directly from the surface of the auricle and of recording the instant at which the electrical disturbance is signalled to arrive at any given point against a standard electrical curve; for the last purpose the electrocardiogram from lead *II* is employed. Working in this way, the relative times at which the excitation wave arrives at different points of the auricular surface, have been ascertained relative to each other with a very small error (0.001 sec.). An even closer result may be obtained by timing the arrival of the excitation wave by means of independent but simultaneous direct leads from the points investigated.

Methods, so far devised, to record the onset of contraction at a given point have been far less exact. The apparatus employed has been heavy, the levers are slow in movement, yielding an unknown but probably considerable delay. A further serious criticism of such apparatus is that it loads the auricular muscle, which no longer contracts against resistances which approach natural resistances. Another defect is the damage, more or less serious, to the delicate tissues, which is almost inevitable in fixing apparatus to the auricular surface. The lightest and most accurate apparatus of which I know is that recently devised by Wiggers³ for taking curves from single muscle strips, the records being taken optically.

It seemed at the outset, however, that none of the available apparatus would meet the needs of the present observations, which require that the onset of contraction be signalled at different surface points in series with a usual error considerably less than 0.01 of a second.

Method.

The apparatus finally adopted is a relatively simple device, which can be constructed and set up in any laboratory. It consists essentially in a grid of fine and flexible fibres. Human hair, if fine and straight, suits the purpose well; fine strands of silk such as may be obtained by unweaving Japanese silk or the French "peau de soie" is even better.*

It is essential that there should be little wiriness in the thread, otherwise slight up and down movements may be translated into lateral movement. The threads should be approximately 20 centimetres long. To one end of each thread a tiny bead is tied, care being taken that the surplus end of the thread is cut off as short as possible to the knot;† the other end is threaded through a very fine needle and tied to it by a single knot. The grid is now to be constructed.

Dogs of 8 to 12 kilogrammes body weight have been the subjects of the experiments. They have been fully anæsthetised with morphia, paraldehyde and ether. The chest is opened by splitting the sternum, the side walls being held well back and secured; the pericardium is opened over the right auricle and stitched to the chest wall.

A minute portion of the epicardium is caught up on the point of the needle and the thread and fibre are drawn through until the bead is reached; the latter now lies on the surface of the auricle. The thread is suspended over a grooved bar (a screw thread answers the purpose well, see Fig. 1, 1st bar). The thread is now carried over a second and similar bar, placed a little below and to the far side of the first, and a small cylinder of moulders' clay is driven on to the needle point to weight it. The remaining threads are similarly arranged. The needles are driven through the epicardium as equidistantly as possible and along a single line, such as that of the right appendix or that of the *tænia terminalis*. It is surprising how little of the epicardium need be caught up to secure the bead permanently in place. The threads are carried over grooves in the 1st bar so that they shall hang vertically and more or less parallel to each other (or they may incline together above). The purpose of the 2nd bar is to keep the clay weights apart and to maintain them at a safe distance from the grid.‡

The record is taken by projecting the shadows of the vertical threads on to the horizontal slit of a moving plate camera such as is used in electrocardiography.

In the present observations the grid has been interposed between the eye piece of the galvanometer and the camera. In this way a somewhat

* If silk is employed, it should be lightly oiled, otherwise it absorbs blood, which stiffens the thread when it dries.

† In using hair, which is relatively wiry, this precaution is very important.

‡ Accurate curves may also be obtained by dispensing with the beads and fixing all the threads at their bases to the spiral turns of a light spring, the grid being then brought to rest against the edge of the appendix. This method was used in the earliest attempts. It avoids tampering with the epicardium, but is less reliable, as the threads are apt to slip to and fro on the appendix during its movements. Obviously it is inapplicable to the *tænia*.

magnified image of the grid is projected, the magnification varying between 1.3 and 2.3.

To obtain a goodly percentage of successful records certain precautions are necessary. In recording the movements of the threads along a horizontal slit, only those small segments of the thread-shadows are photographed which

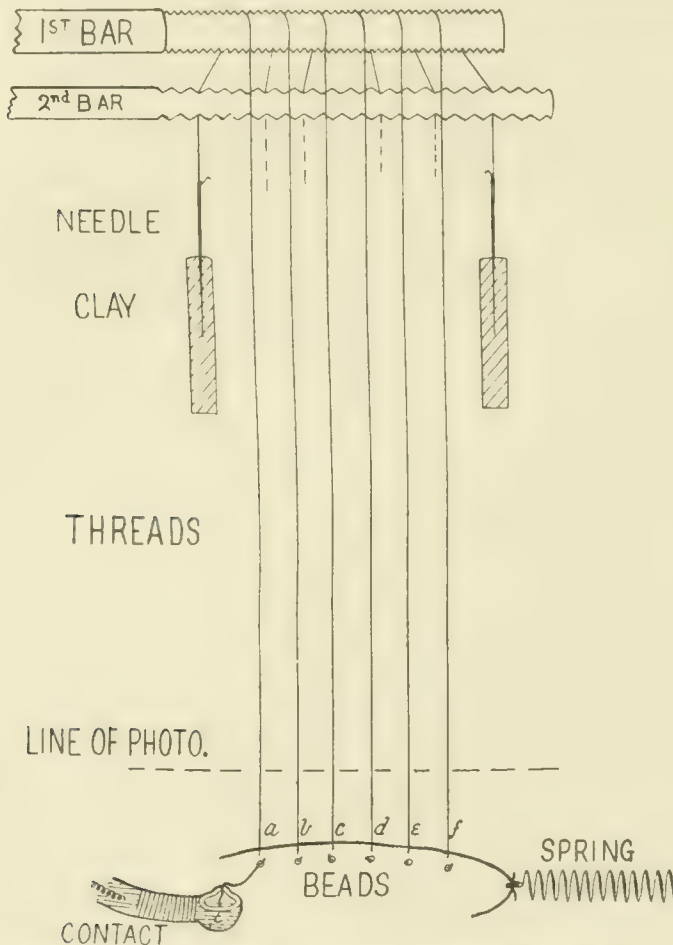


Fig. 1. The polynomyograph, natural size. Six threads (*a-f*) are shown attached by means of small beads to the auricular appendix, the tip of which is lightly balanced by a spring. The threads pass over grooved bars, and the needles are weighted with cylinders of clay. For the sake of simplicity only two needles with the attached weights are shown. The line of the photograph is indicated by a broken line crossing the threads at right angles. A non-polarisable contact is shown, attached to lead *a*. As a rule two such contacts are used, the one attached to bead *a*, the other to bead *e*.

actually fall on the slit. The resulting curves are the more accurate, the nearer the section of the grid photographed (line of photo. Fig. 1) approaches to the beads. The distance should not exceed 1 or 2 centimetres. The line of the strip of muscle investigated should lie as parallel to the camera slit

as possible, that is to say, it should be brought to the horizontal. The counter weights on the threads should be light, so that the auricular tissue is pulled upon to the least extent. At the same time weight is necessary to give the threads a little tension. The threads must have sufficient tension to be straight; with insufficient tension they tend to vibrate. There should be little or no up and down movement of the weights, for up and down movement tends to be converted into lateral movement from time to time. As a rule, weights of from 0.7 to 1.0 grammes are utilised. This weight does not fall wholly on the thread; a good deal of it is taken up by the friction of the bars.*

To pull a single hair of the grid horizontally through 1 centimetre requires, so we calculate, a maximum weight of 0.05 of a gramme.

In working upon the right appendix a light spring, attached to the tip of the appendix, is necessary. Its object is not to increase the excursion of the threads but to help maintain the alignment of the tip of the appendix in its contraction and relaxation. A very light spring is sufficient, though the most favourable tension† varies in different experiments. In working upon the *tænia* a spring of this kind is not necessary.

The records of the grid may be taken simultaneously with electrocardiograms from limb leads (Fig. 2). They may be taken simultaneously with direct leads from the muscle (Fig. 6, 7 and 9). In the last case a special procedure is adopted. A fine strand of cotton is threaded through and tied to one of the beads‡ which lies on the auricle; the free end of the cotton strand is immersed in a small cup of normal saline, which cup forms the end of a non-polarisable electrode (see Fig. 1, *contact*). This strand of cotton, kept moist by capillary attraction, forms one contact; it is connected to the galvanometer in such a way that when it becomes relatively negative, an upright deflection is yielded; it is paired with an indifferent non-polarisable contact on the body wall. In using direct leads and recording the corresponding excitation waves simultaneously with the movement of the grid, it has been the habit to employ two such leads together; selecting for one contact the bead which lies proximal to the pace-maker (Fig. 1 *a*) and pairing it with a body contact, and selecting the most distal bead but one (Fig. 1 *e*) and similarly pairing it with a body contact. This selection gives records of the arrival of the excitation wave at points marked by beads *a* and *e*.

Curves of the grid. These curves are magnified records of the lateral movement of the threads. Each thread inscribes a curve on the plate which corresponds to the movement of the point of muscle to which it is attached. Consider the beads of Fig. 1. When that portion of the base

* Small pulley wheels have been used and have been discarded as less favourable, they yield more up and down movement and tend to override. The frictional suspension helps to fix the upper end of the thread, which has sufficient elasticity to take up a little up and down movement.

† A tension equal approximately to 2 grammes during systole is usually employed.

‡ The beads are selected and threaded before the grid is constructed.

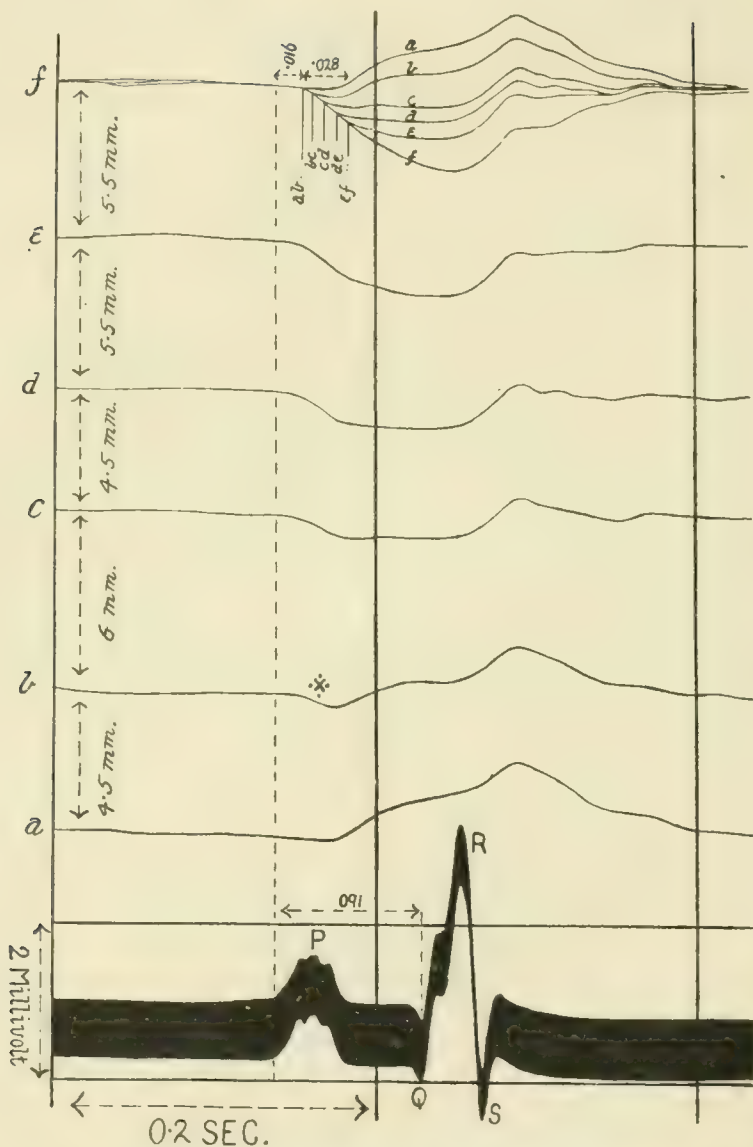


Fig. 2. Dog KC. Record 17. A tracing (reduced to half size), from an enlargement of the original negative, a print of which is shown in Fig. 13. The electrocardiogram (from lead *II*) and six thread curves (*a* to *f*), taken from the ténia terminals, are depicted in their original relations to each other; curves *a* to *e* have also been carried up and superimposed upon the top curve *f*, so that the points at which contraction begins in the muscle segments *ab*, *bc*, etc., may be displayed. The actual diastolic lengths of these muscle segments are indicated to the left of the chart in millimetres. The *P R* interval measures 0.091 of a second; the interval between the beginning of *P* and the first sign of contraction in muscle segment *ab* is 0.016 of a second, and between the latter and the first sign of contraction in *c* it is 0.028 of a second. Three time lines are shown. A photograph of the corresponding auricle is shown in Fig. 11.

of the appendix, which lies to the left of bead *a* in the figure, contracts, it pulls all the beads *a* to *f* equally* to the left. All the threads simultaneously record this initial movement of the auricle. When the strip of muscle between beads *a* and *b* contracts, these beads *come closer together*; contraction of the same muscle strip pulls beads *c* to *f* equally towards the left. As bead *f* lies nearest the free tip of the appendix, its movement will be greatest, for it is pulled to the left by the contraction of all strips from the base to the apex of the appendix; the movement of bead *a* will be least, for it is pulled to the left only by that section of muscle which lies between it and the sulcus terminalis. The degree of movement of beads *b* to *e* will be intermediate and graduated.

In the actual records (Figs. 13-17) of the grid movement, the *beginning of contraction in the muscle covered by the grid is not signalled by the lateral displacement of a thread.†* but by the drawing together of a pair of adjacent threads. Threads *a* and *b* begin to approach each other when the muscle between them begins to contract; they may both move, maintaining, however, their distance from each other, at an earlier instant in response to the contraction of outlying muscle.

The beginnings of contraction are rarely to be seen by inspection of the records; these require analysis.

Analysis of the records. Several methods of analysis have been used, but the most satisfactory is undertaken in enlargements from the original plates. The enlargement is made on smooth bromide paper to four diameters, and the curves are traced with a sharp pencil on oiled tracing paper. Using a good lens for enlarging, the error arising from distortion is inappreciable. A source of error which should be watched, but which should also be inappreciable, results from uneven shrinking of the enlarged print while it dries. Fig. 2 may be used as a convenient illustration of the method. A print from the original plate is shown in Fig. 13. The record is of six threads attached along the length of the *tænia terminalis*,‡ and of a simultaneous electrocardiogram from lead *II*. A portion of this plate has been enlarged and the tracing from the enlargement is shown in Fig. 2. In this and all other figures of this paper, the threads and their corresponding curves are named *a*, *b*, *c*, etc., the lettering starting in each case at the thread which lies nearest to the cephalic end of the *tænia terminalis* or nearest the base of the right appendix. In Fig. 2, the curves inscribed by the six threads *a* to *f* are labelled on the edge of chart: *a* corresponds to the cephalic or superior caval end of the *tænia*, *f* corresponds to the caudal or inferior caval end of the *tænia*. The actual distances of the threads from each other in

* I say "equally," for that is so in general; exceptions will be spoken of later.

† In the reproductions this actual lateral movement is expressed, of course, by up and down movement, as it is in electrocardiography.

‡ The actual points of attachment in this experiment are shown, relative to the muscle bands, in Fig. 11.

auricular diastole are shown to the left of the figure in millimetres. The six curves run at first almost horizontally; this is the period, as seen from the simultaneous electrocardiogram, which immediately precedes auricular systole. The curves in this phase are parallel to each other. Shortly after the appearance of *P*, the representative of auricular systole in the electrocardiogram, the thread curves are displaced. The displacement of the central thread *c* is least, for it is attached to the centre of the tænia; the threads to the superior and inferior caval sides of this central thread are drawn towards the central thread in varying degrees. To estimate the degree to which the threads approach each other, and the instants at which the contractions begin, the curves are superimposed. The resultant series of curves is shown at the top of Fig. 2. The curves are brought together until the horizontal stretches of curve which precede auricular systole coincide. Thus the curves are brought together in the period of diastole, the period during which they lie farthest apart. Subsequent separation of the superimposed lines indicates contraction. Thus in the case of the lowermost curves, *a* and *b*, the approximation of the two threads begins where curve *b* dips towards *a* (marked by an asterisk in Fig. 2); the asterisk marks the beginning of contraction in the muscle strip *ab*. This point is brought out sharply by superimposing the two curves in the manner described and is indicated by the vertical index mark *ab* at the top of the diagram. The remaining index marks, *bc*, *cd*, *de*, and *ef*, mark the points at which contraction begins in the remaining and corresponding strips of muscle.

These index marks demonstrate the order of contraction in the five adjoining strips of muscle investigated, and in this instance it is an orderly sequence starting at the cephalic and ending at the caudal extremity of the tænia terminalis.

Preservation of landmarks. At the end of the experiment the distances between the beads is measured in situ while the auricle is in diastole. Each bead is caught in forceps and the corresponding thread is pulled back a little; the thread is now tied into the auricle at its original point of attachment and cut short. The heart is removed, washed and distended with a solution of formalin and eventually carried into spirit. The hearts so preserved form a permanent and exact reference for the muscle points investigated. They are carried into xylol and cleared, when a more distinct view and photographs (Figs. 11 and 12) of the muscle bands are desired.

RESULTS.

The sequence of contraction in the tænia and right appendix.

The following observations are based upon experiments on eleven animals. In one of these, a series of thread curves was taken from the tænia only, in seven from the right appendix only, and in three from both the tænia and right appendix. The statements are based therefore on a

large collection of curves. In reviewing these curves as a whole and selecting examples which accurately represent the contraction events in the muscle regions investigated, certain curves are to be eliminated. The reasons for so eliminating them are now to be stated.

1. In a small proportion of records the thread curves are found to be parallel over no appreciable part of their course. In such, a safe base line for superimposition cannot be obtained. The best base lines are obtained when the action of the heart is relatively slow, so that movements of the ventricle in its systole or early diastole do not overlap the auricular systole; and when the movements of the ventricle are not jerky. Smooth working of the ventricle is obtained most readily by maintaining that organ so far as possible within its pericardium.

2. In a small proportion of records, although the curves are parallel in the phase preceding auricular systole, the value of the base line is destroyed at the time when auricular contraction begins. There is at this instant a preliminary widening of the gap between one or perhaps more than one pair of adjacent threads. This widening, while it is to be interpreted as resulting from actual lengthening of the corresponding muscle strip, is not to be interpreted as resulting from its active relaxation. It is due, presumably, to the pull which one strip in contracting exerts on its as yet passive neighbour. It is to be seen more especially in curves taken from the appendix, with the appendix pulling against a spring attached to its tip; it is largely because of this effect that no considerable countertension is justifiable or indeed compatible with success. The appearance of this preliminary relaxation and the difficulty which it introduces in estimating the onset of contraction is illustrated by Fig. 3. This diagram is a tracing from a record of four threads (*a* to *d*) set into the appendix. Contraction is first shown by strip *ab*. In the strip *bc*, *relaxation* begins at *bc 1*, simultaneously with contraction in *ab*, and is signalled by the preliminary rise of curve *b* above that of curve *c* in the superimposed portion of the diagram. At *bc 2* curves *b* and *c* meet again and cross, indicating that the muscle strip *bc* has now assumed its original diastolic length, and from this point onwards the strip continues to contract. It appears to me to be uncertain whether the active contraction of *bc* is to be taken as beginning at *bc 2* or at a point intermediate between *bc 1* and *bc 2*: the latter is perhaps the more probable.

That lengthening of an auricular muscle strip may occur apart from its actual relaxation has been concluded already by Wiggers,³ who rightly points out that, however ideal the system of recording may be, absolutely reliable mechanical records can never be obtained of the state of muscular activity. The special relation of any two points on the auricular muscle surface is not governed exclusively by the active contraction and relaxation of the muscle strip which lies between them.

In some of my records it is clear that contraction of the ventricle affects the length of the auricular muscle: it is also clear that different strips, both of the tænia and of the appendix (the latter held by a spring) are affected by this ventricular contraction in different degrees (*vide infra*).

Stretching of an auricular muscle strip, through the contraction of a neighbouring auricular strip, will always appear and be recognised in thread

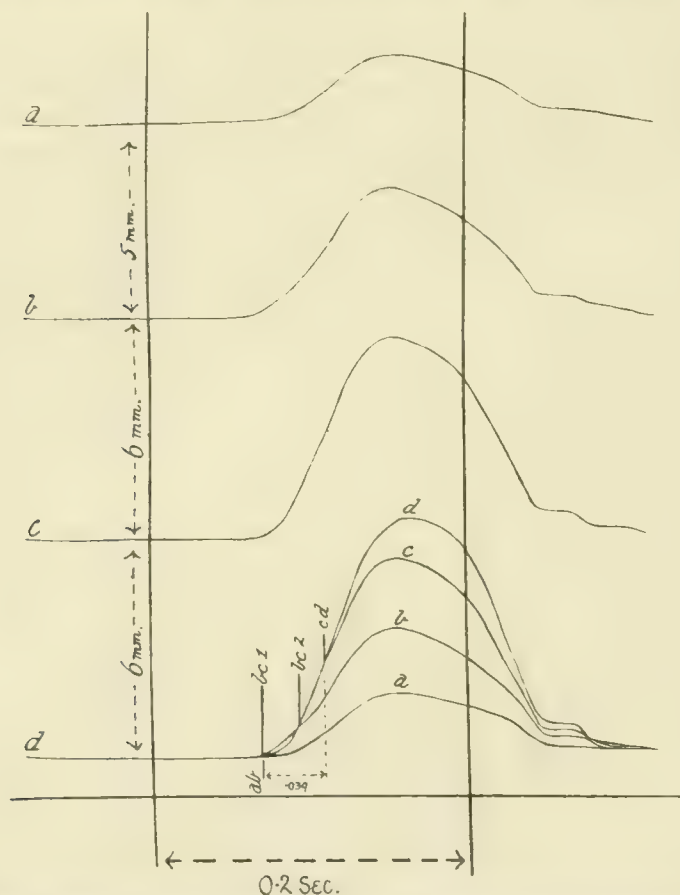


Fig. 3 Dog K.J. Record 6. A similar tracing to the last; reproduced at half its original size. Four thread curves (*a* to *d*) from the right auricular appendix are shown, and curves *a* to *c* have also been superimposed upon *d*. The tracing illustrates a series of curves from which the onsets of contraction in the muscle segments cannot be judged accurately, owing to relaxation of one of the muscle strips (*bc*) immediately before its contraction. As a result curve *b* crosses curves *c* and *d*. Two time lines are shown.

curves providing that it precedes the actual contraction in the same strip. Doubtless the two processes are occasionally synchronous: in such a case a small error would unquestionably be introduced in calculating the beginning of active contraction, the apparent beginning of contraction being delayed. But if the process is present to an appreciable extent it is most unlikely

that it will fail to display itself in any one of the series of strips in the form of preliminary relaxation. The elimination of all curves demonstrating a preliminary relaxation of any strip seems to me a sufficient safeguard.

3. The third ground in which records are eliminated is the presence of relatively insufficient shortening between any two adjacent threads. Fig. 4, taken from the same experiment as the last illustration, serves to emphasise this point. The amount of shortening in strip *cd* is small: the two curves *c* and *d* rise together in the same slope and separate very gradually. The point at which contraction seems to begin is ill-defined, and also tends to be delayed (compare with Fig. 3, in which for strip *cd* there is probably also a slight delay). Such curves are evidently far less safe to use in measurement than such an example as that shown in Fig. 5, in which the obtuseness of the angles where the curves separate is striking.

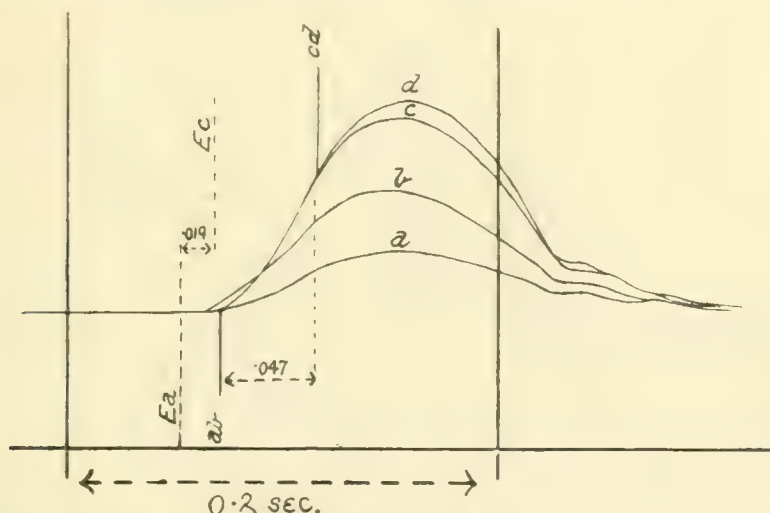


Fig. 4. Dog KJ. Record 7. Portion of a similar tracing from the same animal, reproduced at two-thirds its original size. It illustrates the same defect as the last figure, *viz.*, crossing of the lines. It also illustrates a second defect, namely, insufficient shortening of muscle segment *cd*: this insufficient shortening renders the first apparent sign of contraction difficult to fix and delays it. The times at which the excitation waves appeared at beads *a* and *c* are shown by the index lines *Ea* and *Ec*. There is no correspondence between the transmission interval of the excitation wave (0.019 of a second) and of the apparent transmission time of the contraction wave (0.047 of a second). Two time lines are shown.

The two effects, the crossing of curves dealt with in the last section and the insufficient shortening here illustrated, are not uncommonly associated (as in Fig. 4) and are perhaps to be traced to a common cause.

Having eliminated curves on the grounds stated we are left with the great majority of the curves.

The general order of contraction in the tænia terminalis and the right auricular appendix is constant from animal to animal and from moment to moment in the same animal under the usual conditions of experiment.

The contraction wave courses from the superior to the inferior caval end of the tænia and from the base to the tip of the appendix. To these statements there are no exceptions in the curves. In all the curves of most animals, in some curves from all animals, *a regular sequence of contraction from strip to strip is manifest*. Figs. 2, 5 and 6 illustrate this sequence in the tænia, and Figs. 7, 8 and 9 in the appendix. In the remaining curves there are exceptions in detail. Thus, using the notation *ab, bc, cd*, etc., for the muscle strips, in which *ab* represents the strip nearest to the superior cava, the usual order of contraction is *ab, bc, cd, de, ef*. In exceptional cases it may be *ab, cd, bc, de, ef*, one strip showing the onset of contraction out of place in the sequence. Less commonly two strips may show this lack of sequence, (*ab, cd, bc, ef, de*) or one strip may be further displaced (*ab, de, bc, cd, ef*). Where such minor displacements occur they are inconstant, varying not

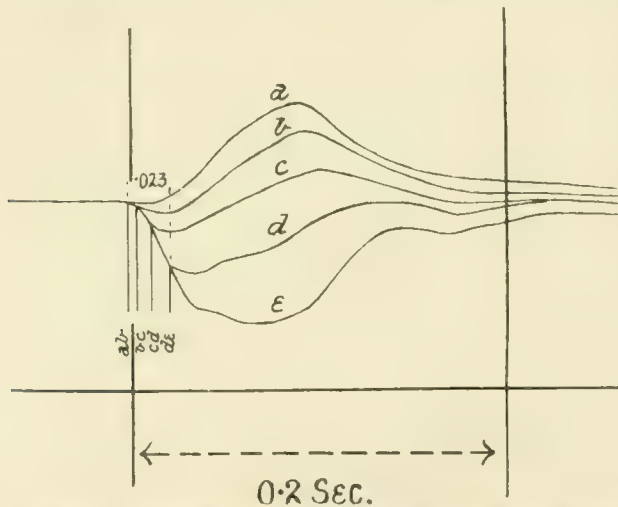


Fig. 5. Dog KE. Record 6. Portion of a similar tracing reproduced at two thirds its original size. The superimposed curves of five threads, set into the tænia terminalis, are shown. The muscle segments of strips *ab, bc*, etc., measured 5, 5, 7, and 7 millimetres in diastole. The transmission interval from thread *a* to *d* equals 0.023 of a second. The curves separate at the contraction of the muscle segments at somewhat unusually oblique angles. Individual curves (*c* and *d*) also display another somewhat unusual feature, namely, a conspicuous alteration of direction when they separate from their neighbours (*d* and *e*). In other words curves *c* and *d* alter sharply at the points where the contraction wave passes the corresponding threads. A similar change of direction is seen in curve *e*.

only from plate to plate, but even on one and the same plate. When it is recognised that the time interval between the onsets of contractions in two adjacent strips of muscle, each approximately 5 millimetres in length, approaches the small quantities such as 0.005 to 0.01 of a second, this departure in detail from a completely regular sequence is not remarkable. The actual error introduced by the apparatus is not precisely known; that it is usually less than 0.005 of a second is clear, that it is often a good deal less than this quantity is also clear; that it may sometimes mount to 0.01 of a second, a

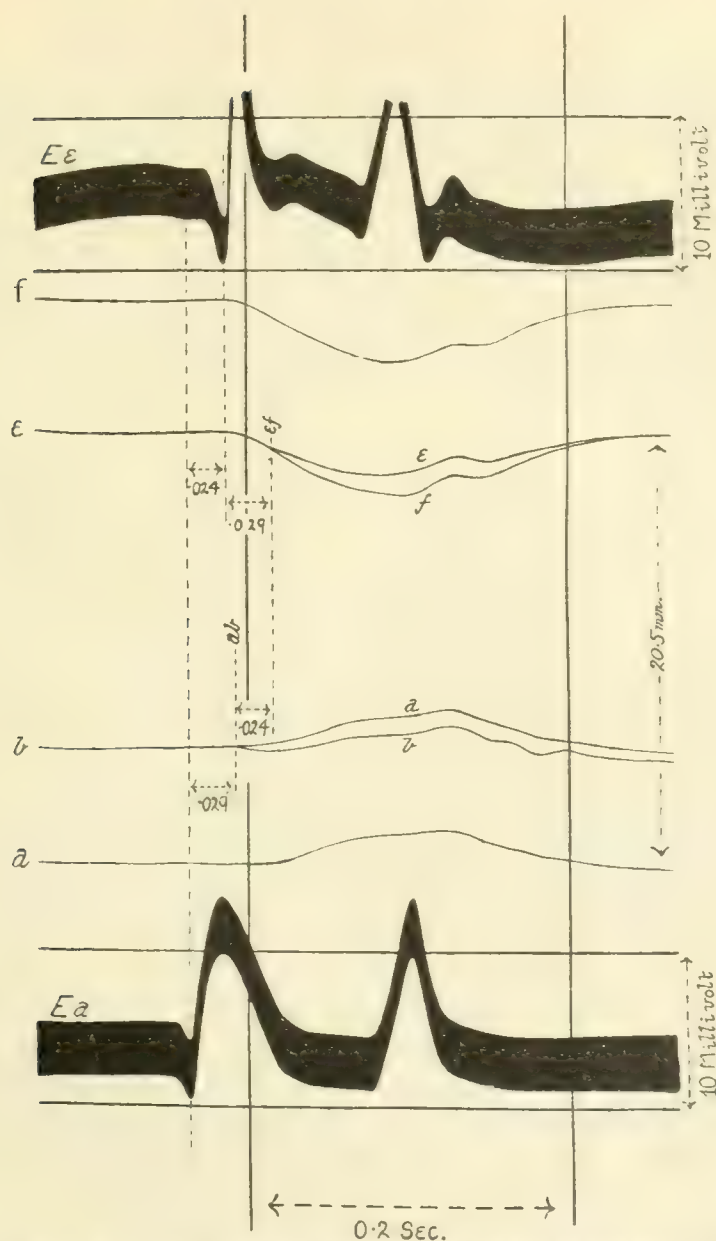


Fig. 6. Dog KC. Record 20. Thread curves from the same grid as that used in the observation shown in Fig. 2; a tracing reproduced at half its original size. The central threads c and d were removed during the experiment, so that the two electrograms Ea and $E\epsilon$ might find room on the lower and upper edges of the plate; these curves were taken by direct leads from beads a and ϵ , and they signal the arrival of the excitation wave at these two beads. Curve a has been superimposed on b and curve ϵ upon curve e ; thereby the times at which the contraction wave arrived at a and ϵ are displayed. The interval between the two excitations is 0.024 of a second, between the two contractions it is 0.024 of a second; the interval between the excitation and contraction is at both points 0.029 of a second.

quantity sufficient to yield the apparent disorder of detailed sequence, is quite probable.

Reviewing the curves as a whole, and remembering the sources of error to which they are open, there is no doubt as to their meaning; the contraction wave spreads as an ordered affair from strip to strip, and it proceeds in a radial direction away from the cephalic end of the sulcus terminalis. We may, I think, without hesitation regard those curves in which the sequence is quite orderly as the most perfect illustrations of the events of contraction; and, comparing them with previous observations upon the course of the excitation wave, we may conclude that *the paths taken by the excitation and by the contraction waves are identical in the mammalian auricle*. This conclusion rests on an examination of two separate regions of the right auricular musculature.

Comparison of time relations of excitation and contraction wave.

The general method by which the transmission interval, of the excitation wave on the one hand and the contraction wave on the other, is compared has been described already. Direct leads from the point of attachment of the threads signal the arrival of the excitation wave. The leads which are used for this purpose are the first of the series (*a*) and the last but one (*e*, where a series of threads *a* to *f* is employed). The grid signals the first contraction in the strips *ab* and *ef*; since the contraction is travelling from *a* to *f*, it signals in reality the arrival of the contraction wave at beads *a* and *e*, the arrival of the excitation wave at the same points is simultaneously recorded. For observations of this kind, only such grid curves are accepted as show a regular and orderly sequence in the contraction of the muscle strips, these being considered, for reasons previously stated, to be the most reliable.

A series of threads (*a* to *f*) was attached to the tænia in the experiment, of which Fig. 2 is an illustration. In the same animal the curves given by these threads were compared with electrograms from points *a* and *e* (Fig. 6, *Ea* and *Ee* respectively). In the tracing, curve *a* has been superimposed upon curve *b* and curve *f* upon curve *e*, so as to display the onsets of contraction in the two muscle strips *ab* and *ef*. The interval between the broken index lines marked *ab* and *ef* in the chart is 0.024 of a second. The electrograms from points *a* and *e* show the moments at which these points become negative quite sharply. The arrival of the excitation wave is signalled by the first upstroke in each curve* and the interval between the two upstrokes is identical with that between the contraction waves (*i.e.*, 0.024 of a second).

* The second upstrokes signal the ventricular activity.

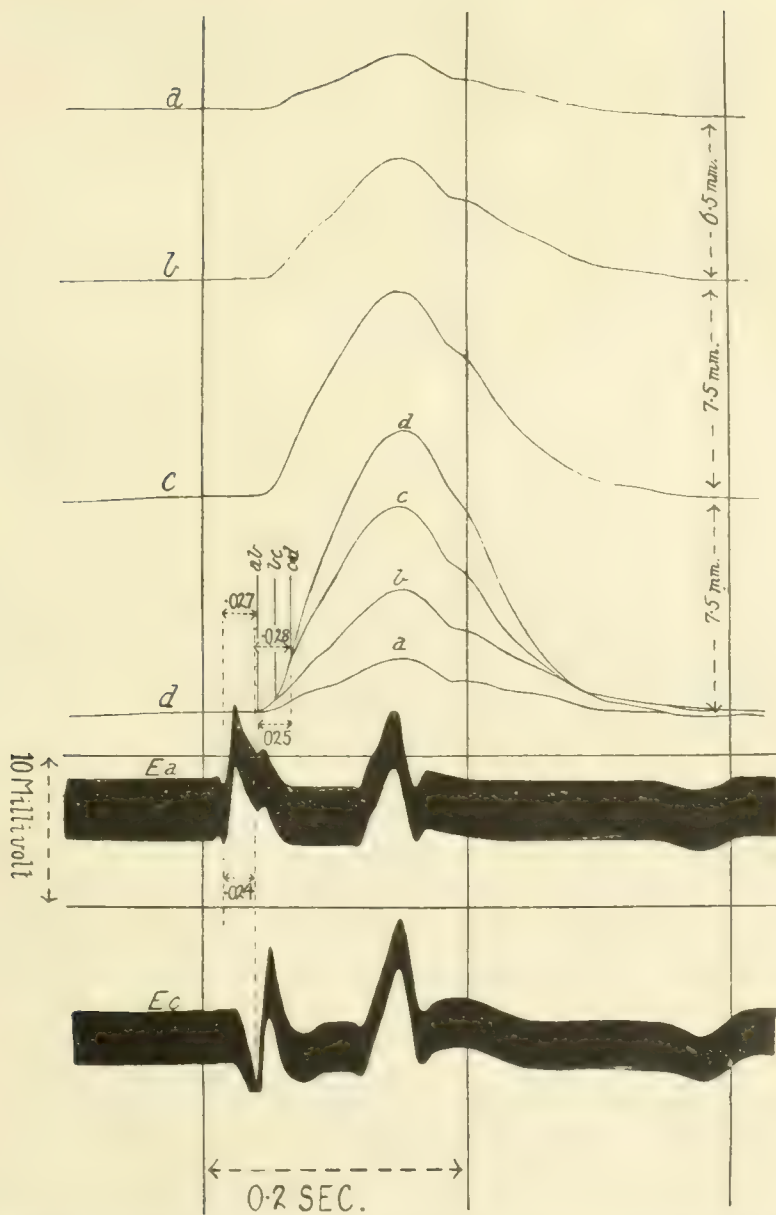


Fig. 7. Dog KK. Record 10. Tracing of four thread curves and two electrograms by direct leads from points *a* and *c*; reproduced at half the original size. The threads were attached to the right auricular appendix. The intervals between the two excitations and between the two contractions are 0.027 and 0.025 of a second respectively. The intervals between the arrival of the excitation and contraction waves at points *a* and *c* are 0.027 and 0.028 of a second respectively.

A second example, illustrating a similar experiment upon the appendix of another animal, is shown in Fig. 7. Here again the transmission interval for contraction wave and excitation wave is the same, namely, 0.025 and 0.024 of a second. These and other examples are shown in Table I, and warrant the conclusion that the excitation and contraction waves travel at precisely the same rates.

From the same series of observations, the time interval between the passage of the excitation wave and contraction wave may be calculated. In Fig. 6 this measurement can be made for upper and lower reaches of the tænia: it is the same for both regions, namely, 0.029 of a second. In Fig. 7 we have also two measurements of the same interval, the one for the base and the other for the apex of the appendix: the intervals are similar, namely 0.027 and 0.028 of a second. The full series is given in Table II.

The usual interval between the appearance of the excitation and contraction waves may be taken for the mammalian heart, beating under ordinary experimental conditions, at 0.02 of a second. Exceptionally it may appear to be as high as 0.05 or 0.06 second.*

The curve of contraction, etc.

The analysis of the thread curve, in so far as the beginnings of the contraction in the segments of muscle are concerned, has now been dealt with fully: but this is not the whole information which such curves yield. Yet the findings in this respect are to be emphasised in that they are more reliable than those which I now propose to describe.

By using a different method of superimposing the curves to that so far applied, a series is obtained which displays different events and which illustrates the action of the grid perhaps more graphically. It consists in bringing the curves together, not until the base lines of diastole meet, but until the curves first touch and coincide. This coincidence first occurs along a short stretch of the curves near the summits of the chief displacements; it represents the phases of the curves during which the threads come closest together and corresponds to the height of contraction in the muscle segments. The first point of actual contact, reading from left to right, represents the end of active contraction; the point where contact is lost again represents the beginning of relaxation, which is then seen to proceed as the curves continue to separate. A tracing illustrating both methods of superimposition is shown in Fig. 8. The grid was composed of threads *a* to *e*, set into the right appendix. The series of superimposed curves at the top is arranged to show the beginnings of contraction; the series of curves superimposed at the bottom shows the endings of the contractions and the beginnings of the relaxations. The events in this tracing are orderly, the contractions

* I do not feel justified in excluding these last values (taken from dog KF), though I am inclined to regard them with some distrust, inasmuch as they depart so far from the remaining values of the table.

TABLE I.

Transmission intervals and rates of conduction of contraction and excitation waves.

Dog.	Record.	Weight in kilos.	Aur. rate.	Region.	Distance in mm.	Trans. time.		Conduction rate.	
						Contract wave in seconds.	Excitation wave in seconds.	Contract wave in mm. per second.	Excitation wave in mm. per second.
KC	19	7.7	108	Tænia	20.5	.019	.018	1080	1139
	20		94	Tænia	20.5	.024	.024	854	854
KF	7	12.0	126	Rt.App.	10.0	.018	.017	556	588
	8		120	Rt.App.	10.0	.018	.015	556	667
	10		108	Rt.App.	10.0	.015	.016	667	625
KG	7	9.9	155	Rt.App.	16.0	.025	.021	640	762
	9		157	Rt.App.	16.0	.023	.021	696	762
KK	9	12.2	104	Rt.App.	14.0	.028	.026	500	538
	10		95	Rt.App.	14.0	.024	.025	583	560
	11		95	Rt.App.	14.0	.023	.025	600	560

TABLE II.

Time intervals between excitation and contraction waves.

Dog.	Record.	Region.	Interval in seconds.
KC	18	Upper tænia	.023
	19	Upper tænia	.022
		Lower tænia	.023
	20	Upper tænia	.029
KF	7	Upper tænia	.029
		Lower tænia	.029
	8	Base of right appendix	.048
		Tip of " "	.049
KG	10	Base of right appendix	.054
		Tip of " "	.057
	7	Base of right appendix	.049
		Tip of " "	.048
KK	9	Base of right appendix	.018
		Tip of " "	.022
	9	Base of right appendix	.018
		Tip of " "	.020
KK	9	Base of right appendix	.017
		Tip of " "	.019
	10	Base of right appendix	.028
		Tip of " "	.027
	11	Base of right appendix	.025
		Tip of " "	.023

it begins, the lines run together more acutely and the margin of error is consequently increased. In the second place, the ends of contraction in distal segments fall at or immediately before the beginning of contraction in the ventricle, and the lengths of the strips may consequently be subject to disturbance at this time phase. Yet, that the ventricle is not often the cause of the disturbance of the ends of contraction seems clear from a more detailed study of the events, and by the observation that imperfection in detail is seen to much the same degree in curves taken from the auricle after the production of heart-block.

Precisely similar reasons may be brought forward to explain the inconstant order in which the relaxations begin, for relaxation is gradual, and, when the heart is beating normally, the relaxations of distal segments of auricular muscle always come at a time when disturbances in the auricle are expected from contraction of the ventricle.

Although the very earliest sign of ventricular contraction is estimated to occur at or near the combined summits of the superimposed curves (*cric. intra*), yet the pull on the auricular muscle resulting from ventricular contraction, seems in most cases to be delayed beyond this. What is often the main effect occurs at the relaxation of the ventricle (see Wiggers also); the auricular segments are pulled upon more or less equally and all the thread curves are raised to a blunt summit near the end of their main descent (auricular relaxation). These are very clearly to be seen in the tracing (Fig. 8) where the ventricular hump in curve *e* is marked *Vs3*. The first sign of pull comes earlier than this, however; it consists of an insignificant summit on the shoulder of the main descent (marked *Vs1* on the same curve). In appendix curves both summits indicate a movement of the threads towards the right side of the dog's body. That these two summits result from contraction of the ventricle is evident from Fig. 16. This record was taken from the same heart and grid while the auricles were in fibrillation, and as movement of the auricular part of the heart now leaves but a slight impression on the curves, the ventricular elements are more distinct. The ventricular curve starts with a small summit (*Vs1*), the upstroke of which coincides with the downstroke of *R* in the electrocardiogram. The curve then dips away and is interrupted by a smaller and inconstant summit (*Vs2*) and finally ends with the broader and prominent summit (*Vs3*). The curve as shown by each thread presents much the same general appearance.* In view, however, of the fact that the summit *Vs1* is constantly a little taller in curve *e* than in curve *a*, the ventricle by its contraction is shown to have produced slight shortening of portions of the muscle strip *ae*. Immediately afterwards, there is a slight lengthening of portions of the strip, for the descent is constantly greater in curve *e* than in curve *a*.

A second example is seen in Fig. 17, which was taken from the auricular appendix after complete heart-block had been established in another animal. Analysis of this curve shows again three ventricular waves, *Vs1*, *Vs2* and *Vs3*. *Vs1* is in this instance the most prominent; it begins at a point which corresponds to the end of the *R*'s downstroke. It should be noted further that since *Vs1* is much more prominent in curve *e* than it is in curve *a*, and that since there is a gradual decline in height from *e* to *a*, there has been shortening of all the segments of auricular muscle included in the grid.

The most conspicuous influence of ventricular systole, then, is shortening of the auricular muscle segments, produced presumably by a drawing together of those parts of the auricle which surround or continue into the auriculo-ventricular ring. This statement applies to those portions of the auricle examined; I presume the event might be reversed if a line of muscle from *tenua* to *A-V* ring were examined. It seems clear that the ventricle may lengthen or shorten the auricular muscle segments; the influence of its contraction is complex.

In inferring the influence of the ventricular contraction upon the immediate summit of the whole auricular curve, I speak of an event not witnessed, for the first disturbance actually seen has fallen a little later in time, namely, on the shoulder of the descent. It coincides with relaxation of the segments. In distant segments of the auricular muscle, however, it would sometimes fall with the end of contraction; moreover, it is conceivable that the effect of ventricular contraction is not always as delayed as these examples would seem to indicate. Even if the contraction of the ventricle but rarely interferes with our estimate of the endings of segmental auricular contraction, it is certain that it often disturbs our estimate of the beginning of relaxation.

* In some curves the pull seems to be quite equally conveyed to all threads.

The endings of relaxation in the auricular strips are still more gradual than the beginnings; they are in fact so irregular in the thread records that the attempt to time them has to be abandoned. These endings are very imperfectly represented, for example, in the top series of superimposed curves of Fig. 8, where the curves finally come together to form a common base line; here they are seen to fall simultaneously with the last and main summit which is due to ventricular contraction. As Wiggers has pointed out, not only is active relaxation involved, but so is also the relaxation of auricular distension.

In drawing conclusions as to the points at which segmental contraction ends and segmental relaxation begins, I confine myself to curves in which all the events are orderly, as in Fig. 8.

The time relation between the ends of the segmental contractions and the onset of contraction in the ventricle is worthy of closer study. The usual events may be illustrated by Fig. 9. The analysis of the thread curves is given above and in proper time relation to an electrogram, taken from contacts, the one on head *a* upon the auricular surface, the other upon the left hind-leg of the animal. This curve shows in the first instance a sharp upward deflection *I*, which represents the arrival of the excitation wave at head *a* on the auricular surface. Later, it shows the ventricular complex, consisting of small but quite distinct deflections, *Q*, *R* and *T*. The contraction of muscle strip *cd* ends while *R* is beginning to ascend, it is delayed beyond the opening phase *Q* of the ventricular electrocardiogram by an appreciable interval (0.012 of a second). This is the usual relation in so far as the right appendix is concerned; the strip of muscle near the tip of the appendix ends its contraction as *R* is beginning to rise (or a very little earlier or later).

Now *contraction* in the ventricle, as estimated from records of the first heart sound, begins in the dog 0.01 or less to 0.02 second after the beginning of the upstroke of *R* in the electrocardiogram. Contraction, so estimated, begins in some instances as early on the upstroke of *R* as the junction of its first and middle thirds; in other instances it is delayed to the summit of *R*. Seeing that the muscle record is taken at some little distance, usually about a centimetre from the extreme tip of the appendix (which may be calculated to complete its contraction 0.01 of a second later), we shall not be far wrong in concluding that contraction in the right auricular appendix does not reach its height before contraction in the ventricle is sufficiently under way to throw the auriculo-ventricular valves into tension. The two events are almost simultaneous. But the tip of the right appendix is not the last portion of the whole auricle to enter the state of contraction, if we may judge, as we are now justified in doing, from the time relations of the excitation wave. The tip of the left appendix, the lower parts of the auricular septum, the muscle sleeves on the left pulmonary veins, on the inferior vena cava and on the coronary sinus contract very distinctly later. There are certainly

distant* portions of the muscle, in which contraction begins some 0.02 of a second later than in the tip of the right appendix in dogs of the size employed. While we may conclude therefore that the height of contraction is reached in the main mass of the auricular tissue almost if not quite simultaneously with the first movement of the auriculo-ventricular valves, we must recognise that in the most distant regions of the auricle, and especially in the muscular sleeves of the entering veins and in the A-V rings, it is not quite, though it is almost, at its height at this phase of the heart beat. These time relations demonstrate a nice physical adjustment, the shock of the ventricular movement and the tension on the valves and auriculo-ventricular rings being met by an auricle, whose musculature is largely still at full tension.

Length of fractionate contraction. I use this phrase in the sense of Wiggers, its originator, to indicate the duration of contraction at a single point of the muscle substance. The curve of fractionate contraction is and must remain theoretical; we arrive at a conception of it by studying curves of muscle strips. As Wiggers indicates, curves from such strips are complicated by movement of the contraction wave through the strip while it inscribes its curve. The duration of the rise in a myocardiogram gives neither the length of contraction of the auricle, nor of the fractionate contraction. If it is desired to know the length of auricular contraction then it is necessary to investigate every part of the auricle. Seeing that the first appearance of the excitation wave precedes the beginning of *P* in an axial electrocardiogram, taken at the usual sensitivity, by an average interval of 0.01 of a second, and seeing that the contraction wave succeeds the excitation wave by the usual figure of 0.02 of a second, we may conclude that auricular contraction first starts in the average 0.01 of a second after the beginning of *P*; it ends, as has been calculated, some 0.02 to 0.03 second after the beginning of *R*. Thus, within one or two hundredths of a second, the *P-R* interval may be taken to express the length of auricular contraction, when in dogs of 8 to 12 kilos weight the heart is acting normally, and beating at rates around 100 to 120 per minute. The length of the *P-R* interval in dogs, in these circumstances, lies normally between 0.08 and 0.12 of a second; the length of auricular contraction is 0.09 to 0.14 of a second; the intervals given by Wiggers for his experiments vary between 0.08 and 0.14 of a second.

The fractionate contraction is calculated by subtracting from the whole contraction time of a strip of muscle, the time taken for the contraction wave to pass from one end of the strip to the other. The shorter the strip investigated the more nearly does the whole contraction time estimated for that strip approach to the fractionate contraction time. The duration of contraction in the muscle strips and the fractionate contraction time of the present experiments are exemplified in the accompanying table.

* Distant from the pacemaker, the sino-auricular node.

TABLE III

Dog.	Weight in kilos.	Auricular rate per min.	Average length of contraction in muscle segments in secs.	Average transmission time per segment* in secs.	Fractionate contraction time in secs.
JZ	10.5	116	0.079	0.015	0.064
KF	12.0	121	0.064	0.009	0.055
KG	9.9	153	0.065	0.008	0.057
KK	12.2	98	0.081	0.009	0.072
				Average	0.061*

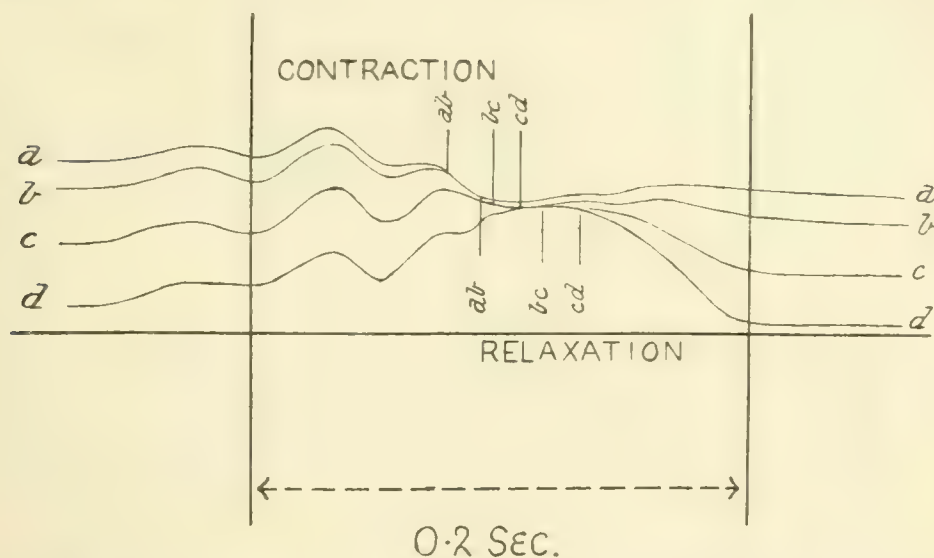


Fig. 10. Dog KG. Record 12. A series of four thread curves from the tænia terminalis is shown. They are superimposed to show the endings of contraction and beginnings of relaxation. In the whole strip (*a'd*) these events overlap. Reproduced from a tracing at two-thirds the original size.

The average length of the fractionate contraction is 0.06 of a second. If the average length of the whole auricular contraction is taken at 0.11 of a second, the difference 0.05 of a second should give us, approximately, the time taken for the contraction wave to spread throughout the entire auricle. This figure agrees well with actual observation of the spread of the excitation wave.

* Segments of 5 to 7 millimetres length.

* These figures are higher than those calculated by Wiggers, whose average works out at 0.047 of a second, and who obtains figures as low as 0.032 and 0.037 of a second. Wiggers' calculations are made by a different method to that here adopted.

Stage of sustained contraction. There is in the curve of auricular contraction a diminutive plateau stage. Its duration averages 0.029 to 0.041 of a second in different animals. Sometimes, in dealing with four or five strips of muscle, each approximately 5 millimetres long, there is no appreciable overlapping of the ends of contraction on the one hand and the beginnings of relaxation on the other; usually some such overlap is distinct (Fig. 10). Clearly, the degree of overlapping depends, not only upon the length of the plateau stage, but chiefly upon the number of strips included in the grid. The absence of an apparent plateau in auricular myocardiograms taken by the usual methods is due to its brevity and to this overlap of contraction and relaxation in closely adjoining segments of the whole muscle strip investigated. In the grid curves the segmental curves and the curve from the whole strip may be compared; the last is built up of the first. Thus, the initial slow ascent of the main contraction curve in its initial phase is seen to be produced chiefly by the gradual involvement of succeeding segments of the muscle strip as a whole, to which cause it has actually been ascribed by Wiggers. Similarly the gradual sweep, falling away in its rate to the summit, is often seen to be in a large measure due to the progressive failure of segmental strips to contract further, rather than to falling away in the rate at which contraction proceeds to its height in the muscle segment.

SUMMARY AND CONCLUSIONS.

A polymyograph is described by means of which accurate curves of muscle shortening and lengthening can be taken from a series of continuous muscle segments, each of approximately 5 or more millimetres length. By means of these curves it has been shown that the order in which the segments of muscle contract in the right auricular appendix and in the tænia terminalis, is the same as the order in which they show the arrival of the excitation process.

By simultaneously recording the curves of contraction and of excitation, it has been shown that the time between the arrival of these two processes, at a given muscle point, is constant at the two ends of a muscle strip, and that contraction and excitation waves travel in the auricular muscle at precisely the same speeds. This observation serves to emphasise the intimate association of the two processes. It is also of value in that it places what has been frequently assumed, namely, that conclusions upon the origin and course of the excitation wave may be read in terms of the contraction wave, upon a firm basis. The interval between the excitation and contraction processes in the dog's auricle, beating at rates of 90 to 150 per minute, is approximately two hundredths of a second.

The fractionate contraction of the auricle, beating in similar circumstances, has a normal duration of 0.06 of a second; this interval, when added to the time which the contraction waves takes to spread throughout the entire

auricular tissue, amounts to some 0.11 of a second or a little more in dogs of 9 to 12 kilos weight. This combined figure gives us the whole period from the beginning of contraction in the first segment to its culmination in the last segment of the muscle. The first event occurs approximately 0.01 of a second after the beginning of *P* in electrocardiograms; the last event coincides with the initial phases of the ventricular complex and extends in distant segments of the auricular muscle a little beyond the beginning of the first heart sound. The segmental contraction is maintained at its height to form a short plateau of 0.02 to 0.04 of a second in duration. Relaxation begins in the main mass of the auricular tissue distinctly after the closure of the auriculo-ventricular valves and is further delayed in the muscular cuffs of the great veins and in the musculature surrounding the *A-V* rings.

The form of a myocardiogram taken from a strip of auricular muscle is modified by the progress of the contraction and relaxation waves through it. The initial rise of the curve is rendered less steep, because the whole strip of muscle is not involved simultaneously; in strips of 25 millimetres length or more the summit reached is a little reduced in amplitude, the rise to the summit is a little flatter, but the actual short plateau is abolished because the endings of contraction and beginnings of relaxation in some segments of the strip coincides with the plateau in other segments.

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Fig. 11. A photograph of the right auricle of Dog KC, a little magnified. The auricle has been fixed, dehydrated and cleared with the threads (*a* to *f*) used for the grid in situ, so that their precise positions, relative to the musculature of the auricle may be seen. The threads have been rendered more distinct by pencilling them. The curves from this auricle are shown in Figs. 2 and 6.

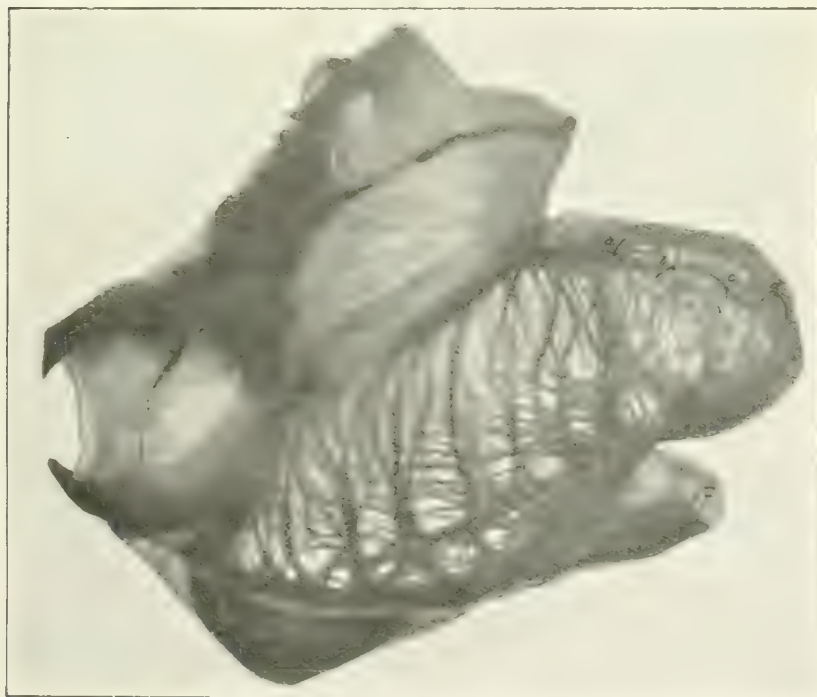


Fig. 12. A similar photograph of the right auricle of Dog KE, a little magnified. The position of the threads *a* to *f* on the auricular appendix are to be seen. The curves from this auricle are shown in Fig. 9.

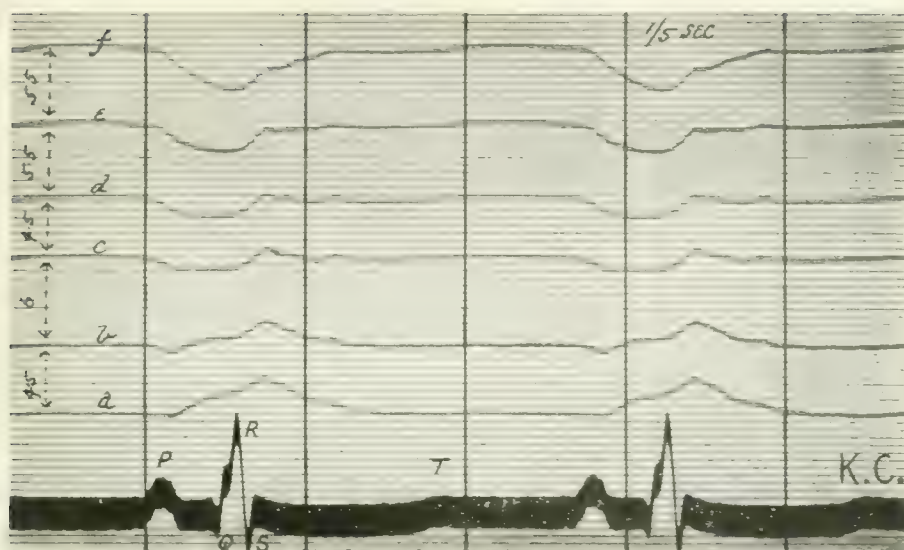


FIG. 13. Dog KC. Record 17. The original photograph, from a part of which after enlargement Fig. 2 was traced. It shows two heart cycles. Above are the six thread curves, obtained from a grid fixed to the tanna tomotals; below is the electrocardiogram from lead II. Time lines in fifths of a second.

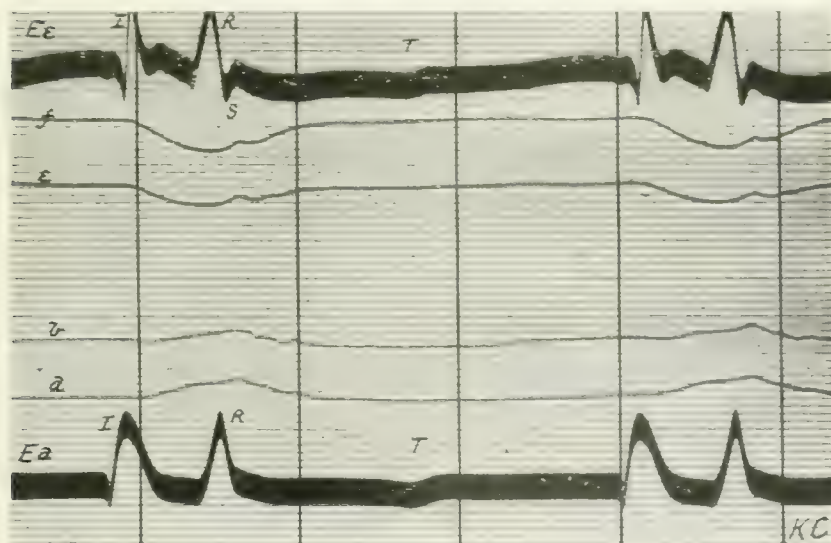


FIG. 14. Dog KC. Record 20. The original photograph, from a part of which after enlargement Fig. 6 was traced. It shows two heart cycles. An electrogram E_e , from bead e , and the corresponding thread curves e and f are shown above; and an electrogram E_a , taken from bead a , and the corresponding thread curves a and b are shown below. Time lines in fifths of a second.

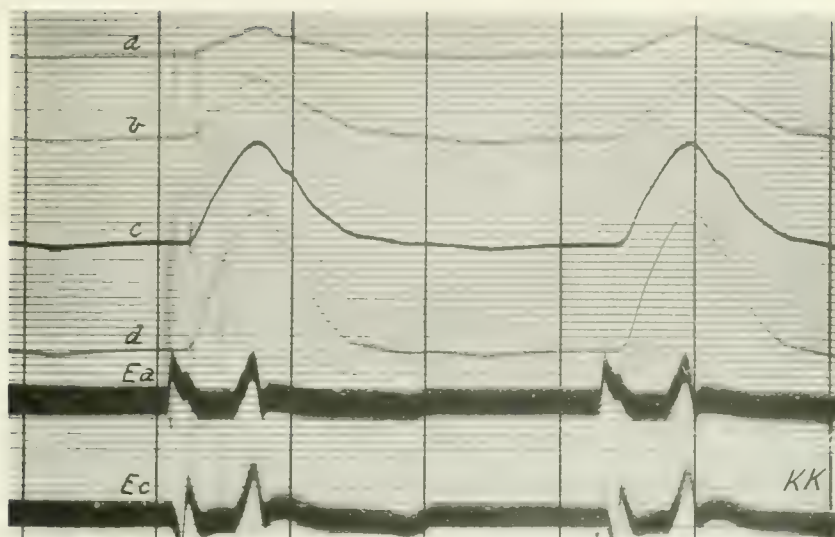


Fig. 15. Dog KK. Record 10. The original photograph, from which after enlargement Fig. 7 was traced. Four curves *a* to *d* taken from the appendix, and two electrograms, *Ea* and *Ec*, taken by direct lead from beads *a* and *c* have been recorded simultaneously. Time lines in fifths of a second.

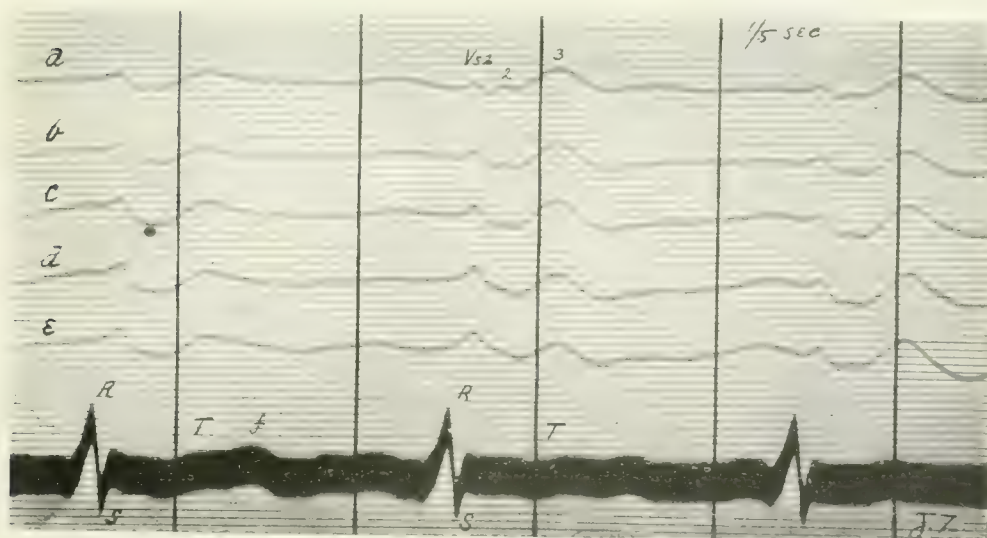


Fig. 16. Dog JZ. Five thread curves from the right appendix and an electrocardiogram from lead *II* are shown. The auricles are fibrillating and the ventricular influence upon the auricle is displayed. Compare with Fig. 8, which is from the same animal. The ventricular summits are marked *Vs1*, *Vs2* and *Vs3*. Time lines in fifths of a second.

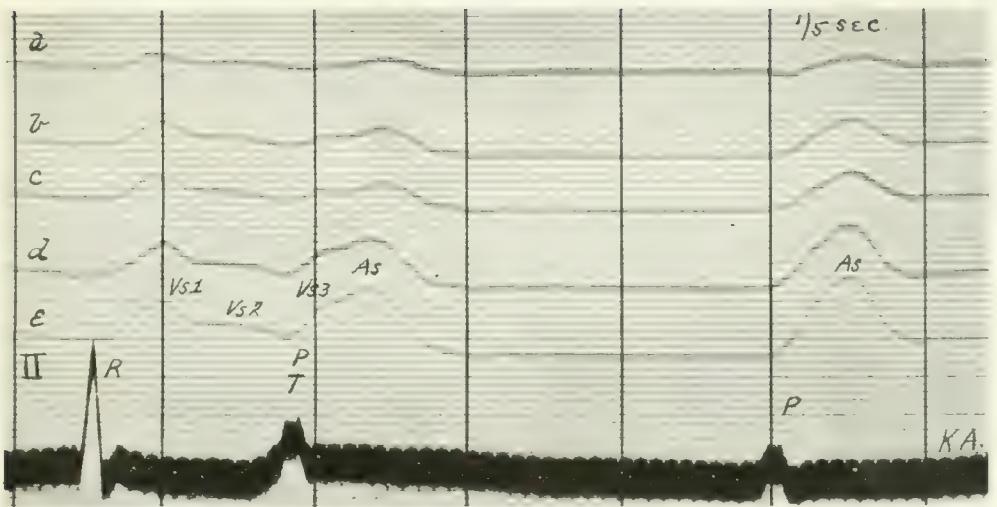
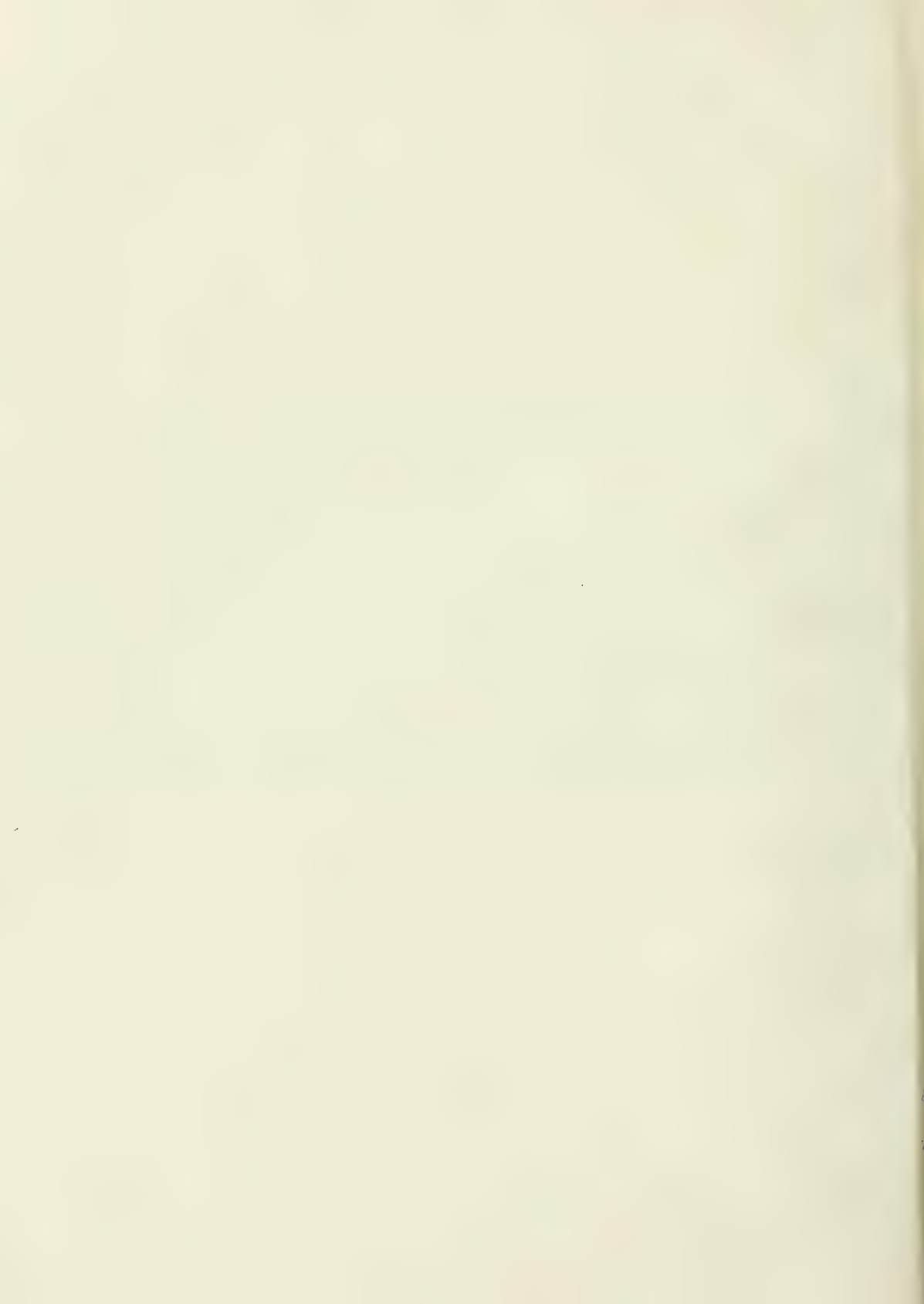


Fig. 17. Dog KA. Five thread curves from the right appendix and an electrocardiogram from lead II, during complete heart block. The ventricular summits on the auricular thread curves are marked Vs1, Vs2 and Vs3. The auricular summits are marked As. Time lines in fifths of a second.



THE PERCENTAGE OF CARBON DIOXIDE IN THE ALVEOLAR
AIR, AND THE TOLERANCE TO ACCUMULATING CARBON
DIOXIDE IN CASES OF SO-CALLED "IRRITABLE HEART"
OF SOLDIERS.*

By ALAN N. DRURY.

(From University College Hospital Medical School.)

THE experimental work was carried out in 1916 at the Hampstead Military Heart Hospital on patients returned to that hospital as cases of "Valvular Disease of the Heart" (V.D.H.), and "Disordered Action of the Heart" (D.A.H.).

All the patients who formed the subjects of experiment were examined by the sorting-out methods¹ then in use at the Hospital, and were considered a representative group of case of "irritable heart."

The experiments were carried out in a room attached to one of the wards. This room was kept at an equable temperature, similar to that of the ward. The only barometer available was an aneroid. This was corrected by comparison with standard instruments and the readings reduced to sea level. The readings have not been reduced to N.T.P., except where stated.

With the instruments available, it was likely that such corrections might introduce even further errors. Variable factors, which cannot be eliminated in experiments on respiration are always creeping in, and mathematical exactitude is impossible.

As the results of the experiments were to be compared with the results obtained from healthy subjects, the chief object was to obtain similar conditions.

The experiments required that the subject should show some intelligence and will power, and as the majority of the patients were of a nervous disposition and showed a marked inability to fix their attention, results were often obtained which could not be considered on account of the patient being unable to do his share of the experiment efficiently.

As a possible guard against this, the patients selected were taught their part of the experiment before the experiments were really undertaken. This enabled much more consistent results to be obtained.

* Undertaken on behalf of the Medical Research Council.

The percentage of carbon dioxide in the alveolar air taken at rest.

The following table shows the results which were obtained from thirty patients.

The method adopted for obtaining the samples of alveolar air was that devised by Haldane.² The patient, after lying down for half an hour,

TABLE I.

Name.	Dyspnœa on mild exercise.	Respiratory rate at rest.	Percentage of CO ₂ .	Average.
1. S. R.	v. sl.	24	5.4, 5.38, 5.51	5.43
2. A. H.	v. sl.	18	5.6, 4.78, 5.38	5.28
3. P. A.	v. sl.	18	5.65, 5.25, 5.38	5.42
4. S. T.	v. sl.	24	5.32, 5.32, 5.52	5.38
5. H. A.	v. sl.	24	5.81	5.81
	Average	21.6		5.46
6. B. W.	sl.	18	5.48, 5.65, 5.81	5.65
7. K. G.	sl.	27	5.5, 5.5, 5.06	5.35
8. E. H.	sl.	20	6.26, 5.07, 5.3, 4.94, 4.58	5.23
9. B. A.	sl.	20	5.6, 5.38, 5.7, 6.0	5.66
10. S. G.	sl.	20	4.86, 5.8	5.33
	Average	21		5.44
11. D. F.	+	20	5.15, 5.13, 4.40, 4.67, 5.0	4.87
12. P. H.	+	20	5.3, 5.1, 4.75, 5.25	5.08
13. G. L.	+	25	4.8, 5.3, 5.01	5.04
14. R. T.	+	20	3.7, 5.05, 5.06	4.60
15. C. J.	+	30	6.2, 5.28, 5.70	5.73
16. H. E.	+	20	4.70, 5.68	5.19
17. S. M.	+	22	5.6, 5.88	5.74
18. G.	+	24	5.78, 5.10	5.44
	Average	22.6		5.21
19. R. L.	++	27	4.26, 4.92, 4.25, 5.20, 4.88	4.69
20. S. T.	++	36	3.74, 3.4, 3.4, 2.97, 3.75, 3.23, 3.4	3.41
21. Jo.	++	30	5.54, 5.28	5.41
22. Pa.	++	30	5.6, 5.1	5.35
23. Pi.	++	39	4.75, 4.59	4.67
24. Mo.	++	36	5.6, 5.2	5.40
25. At.	++	36	5.18, 4.96	5.07
26. E. C.	++	22	5.54, 5.85, 5.0, 5.45, 5.0, 5.1	5.32
27. A. A.	++	28	5.83, 5.14, 4.93	5.30
28. W. R.	++	33	3.79, 4.1, 4.9, 4.65, 4.7, 4.1	4.37
29. S. A.	++	33	5.5, 5.5, 5.37, 5.35	5.43
	Average	31.8		4.94
30. B. R.	+++	40	5.5, 4.82, 5.29	5.21
	Average for Table I	27.4		5.25

placed the mouth-piece in his mouth and after a normal inspiration expired deeply and quickly through the mouth-piece only. The sample thus obtained was a sample of the alveolar air at the end of inspiration.

The apparatus used for the analysis of the sample was similar to that used by Haldane³ for general air analysis.

Barometric pressure was noted. The temperature of the water surrounding the sampling tube was taken.

The percentages of carbon dioxide in the samples have been reduced to N.T.P.

The results obtained show that in thirty patients, giving an average respiration rate of 27 per minute, the average percentage of CO₂ in the alveolar air after a normal inspiration is 5.25. If groups depending upon their reaction to mild exercise are considered, it is seen that slightly higher figures are obtained for those with slight than for those with severe symptoms.

Observations on the healthy resting person, breathing 16 to the minute, show that the percentage of CO₂ in the alveolar air after a normal inspiration varies between 5.54 and 6.17, giving a mean of 5.85.² The figures obtained for the slight cases are within the lower limits of normality; while those obtained for the severer are below the normal figures. The respiration rates, on the other hand, are greatly in excess of the rates of healthy men.

The increase in the rate of respiration is not associated with an increase of CO₂ in the alveolar air.

The percentage of carbon dioxide in alveolar air taken at rest and after an exercise which produced breathlessness.

Twenty-six patients were tested.

Samples of the alveolar air were obtained from the patient after he had rested for half an hour. These were analysed by the method already described. He was then sent on the exercise which consisted of a quick march down and up an incline 130 feet long, rising one foot in seven feet. He immediately came to the alveolar air apparatus. A sample of the alveolar air was taken. After he had rested for four minutes another sample was taken. Both the samples were then analysed.

This gave two readings for each experiment. The first was during the exercise period; the second during the period in which the patient was returning to his pre-exercise condition.

The exercise to which the patients were subjected was of a very mild order. It produced no breathlessness at all in the healthy subject, and very little in the milder cases. It was only when the severe cases attempted the exercise that it had any appearance of being strenuous.

The degree of dyspnoea was noted after the exercise.

TABLE II.

Name.	Dyspnœa after exercise.	%CO ₂ at rest.	%CO ₂ after exercise.	Difference.	%CO ₂ 4 min. later.	Difference from rests.
1. S. T.	v. sl.	5.73	5.92	+0.19	5.76	+0.03
2. B. W.	v. sl.	6.08	6.25	+0.17	5.70	-0.38
3. D. F.	v. sl.	5.35	6.24	+0.89	5.12	-0.23
4. G. L.	v. sl.	5.49	5.24	-0.24	5.60	+0.12
5. K. G.	v. sl.	5.69	5.33	-0.36	5.63	-0.06
6. S. R.	v. sl.	5.81	5.7	-0.11	5.50	-0.31
7. P. A.	v. sl.	5.81	6.01	+0.20	5.84	+0.03
Average		5.70	5.81	+0.11	5.59	-0.12
8. A. H.	sl.	5.56	5.92	+0.36	5.72	+0.16
9. E. H.	sl.	5.61	6.10	+0.49	5.57	-0.04
Average		5.58	6.01	+0.43	5.65	+0.07
10. P. A.	+	5.5	7.10	+1.60	5.90	+0.40
11. B. A.	-	6.03	6.87	+0.84	6.03	±0.00
12. S. M.	+	6.11	5.30	-0.81	5.48	-0.63
Average		5.88	6.42	+0.54	5.80	-0.08
13. R. L.	++	5.08	6.25	+1.17	4.83	-0.25
14. C. J.	++	6.13	7.28	+1.15	5.45	-0.63
15. S. T.	++	3.80	3.25	-0.55	3.64	-0.16
16. Jo.	++	5.80	6.56	+0.76	5.57	-0.23
17. Pa.	++	5.76	6.70	+0.94	5.57	-0.23
18. Pi.	++	5.07	5.74	+0.67	4.84	-0.23
19. Mo.	++	5.88	7.74	+1.86	5.76	-0.12
20. At.	++	5.47	7.10	+1.63	4.99	-0.48
21. Ga.	++	5.88	6.25	+0.37	5.57	-0.31
22. S. A.	++	5.83	4.63	-1.20	5.74	-0.09
23. W. R.	++	4.81	4.86	+0.05	4.25	-0.56
24. A. A.	++	5.69	7.00	+1.31	5.20	-0.49
25. E. C.	++	5.72	6.40	+0.68	5.30	-0.42
Average		5.49	5.84	+0.34	5.12	-0.37
26. B. R.	+++	5.60	5.60	±0.00	4.06	-1.64

Douglas and Haldane¹ showed that in the healthy subject the reaction to exercise depended upon the severity of the work done. If the exercise was of a mild order there was a rise in the alveolar CO₂ pressure during the exercise. This was not followed by any subsequent fall below the normal. The slight and distinct rise which occurs during such an exercise in the alveolar CO₂ pressure is of the order of 0.28 per cent. If, however, the exercise was severe, the initial rise was followed by a secondary fall in the alveolar CO₂ pressure. A sudden and considerable exertion, or a prolonged strenuous exercise would produce such a reaction. The degree of rise and fall in the alveolar CO₂ pressure varied amongst different individuals and in the same subject experimented upon at different times. For a severe exercise there might be an increase of 1.3 per cent. and a fall of 0.5 per cent. The intensity of the reaction depended upon the severity of the work done.

In the preceding table, the irregularity of the figures amongst the milder cases is explicable. The exercise was mild to them and only called forth a slight and uncertain reaction. In the severer cases, where one is dealing with the effects of a strenuous exercise, the figures are much more regular. The reaction is along the same lines as in the healthy individual. There is a primary increase followed by a subsequent decrease in the alveolar CO_2 content. The test exercise required to produce such a reaction is much less severe than that necessary for the healthy subject.

In these cases, as the symptom of breathlessness increased, the intensity of the reaction increased. This was to be expected, as the test exercise to those with marked dyspnoea was of a severe type.

These patients commence with a percentage of CO_2 in the alveolar air within the lower limits of normality or decrease. Unless the reaction is greater than in the healthy subject, the figures obtained during and after the exercise would be correspondingly smaller than in the healthy subject. The reaction in these patients appears to be of the order of the reaction in healthy men to severe exercise.

It can be concluded that the symptoms which appear during and after mild exercise may be accompanied by a lower percentage of CO_2 in the alveolar air than would be met with in the healthy man doing severe exercise.

Assuming that the preceding exercise represented a sharp and sudden exertion to these patients, it is of interest to determine the reaction to a prolonged exertion. With this end in view, a series of determinations was made of the CO_2 content of the alveolar air before and after one of the graduated exercises.* The men were undergoing the routine exercises adopted at the hospital. All the patients tested were tolerating their exercise well, but were distinctly out of breath at the end. The samples of alveolar air were taken immediately before the exercise, and about four minutes after it ceased. It was not practicable at the time to take a sample during the exercise. The results obtained only deal with the period of return to pre-exercise condition.

TABLE III.

Name.	Dyspnoea on mild exercise.	Drill done.	% CO_2 before.	% CO_2 4 minutes later.	Percentage difference.
R. T.	sl.	C. 15*	5.42	4.51	-0.91
		C. 15	5.45	4.75	-0.75
B. A.	sl.	A. 30	6.03	5.45	-0.58
			6.44	5.77	-0.67
G. L.	+	C. 15	5.28	3.82	-1.46
W. R.	++	A. 15	5.38	5.10	-0.28
		A. 15	5.07	4.70	-0.37

* The letter indicates the grade of exercise employed, A being very simple movements and C more severe, and the number the number of minutes.

The figures show a very definite and constant decrease in alveolar CO_2 pressure in the period after the exercise. Haldane and Douglas' found a similar condition in the healthy subject after a prolonged strenuous exertion. This experiment confirms the statement that the reaction to exercise is similar to the healthy person, as far as the alveolar CO_2 pressure is concerned. The test exercises required either for the short and sudden exertion or the prolonged exertion are much less severe than those required for the healthy subject.

The time during which the breath could be held.

Observations were made upon twenty-two patients. A small rubber bag attached to a Mackenzie polygraph was strapped to the patient's chest, so that every respiration was recorded on the polygraph roll. The patients were allowed to breathe quite normally for some time. They were then asked to hold their breath as long as possible. By watching the movements of the pen on the polygraph roll, the patient could be asked to hold "breath"

TABLE IV.

Name.	Dyspnoea on mild exercise.	Resp. per min.	Time breath held in minutes.
1. P. A.	v. sl.	18	11
2. S. R.	v. sl.	24	8
3. H. A.	v. sl.	24	12
4. S. T.	v. sl.	24	10
5. A. H.	v. sl.	18	15
Average		21.6	11.2
6. B. W.	sl.	18	8
7. E. H.	sl.	20	10
8. B. A.	sl.	20	14
9. S. J.	sl.	20	12
10. K. G.	sl.	27	10
Average		21	10.8
11. D. F.	+	20	7
12. P. A.	+	20	11
13. R. T.	+	20	22
14. G. L.	+	25	15
15. C. J.	+	30	7
16. S. M.	+	22	10
Average		22.8	12
17. R. L.	++	27	7
18. E. C.	++	22	10
19. A. A.	++	28	12
20. S. A.	++	33	12
21. W. R.	++	33	9
Average		28.6	10
22. B. R.	+++	40	4

after inspiration or after expiration. The cessation of breathing was recorded by a straight line on the roll. It was often noticed that when the patient was apparently holding his breath there were small excursions of the pen due to his taking small breaths through the nose. These were ruled out. The rate of respiration immediately preceding the "held breath" was noted. The "time during which the breath could be held" was the mean between the times after inspiration and after expiration.

In such an experiment the will power of the subject must affect the result. The point at which a man considers that he can do no more varies amongst healthy men. It is likely to be more variable in a class of patients who are nervous and lack will-power.

Douglas and Haldane¹ found that healthy men could hold their breath for 35-45 seconds.

The average time for these patients was 10.8 seconds, a figure well below that obtained for healthy subjects.

There was no correlation between the rate of respiration and the time the breath could be held. The severity of the symptom of breathlessness did not bear any relation to the "holding breath" period. Douglas and Haldane⁽⁴⁾ consider that two factors are at work, a rise of CO_2 and a fall of oxygen pressure in the alveolus. The experiment shows that the threshold for either of these two stimuli is much lower in all the patients than in health.

*The percentage of carbon dioxide in inspired air which produced
intolerable hyperpnœa.*

Twenty-two patients were tested.

The patients breathed into a mask which covered both the mouth and nose. It was fixed to a threeway tube which was arranged with valves so that the patient could breathe pure air, or in and out of a large bag of 6 litres volume. The patient was seated and told to breathe into the mask as long as he possibly could, even if he had to take quicker and deeper breaths.

At first the patient breathed pure air. After he became used to breathing into the mask, the valves were altered and he breathed in and out of the bag. He continued this until the hyperpnœa produced became intolerable. The bag was then closed with a tap, and the contents analysed for CO_2 by the preceding method.

In order that the time in which the hyperpnœa developed should vary, the bag was filled beforehand with either air, air and CO_2 .

TABLE V.

Name.	Dyspnœa on mild exercise.	Percentage of carbon dioxide.
1. S. R.	v. sl.	6.5, 5.6
2. A. H.	v. sl.	5.6, 5.6, 5.7
3. P. A.	v. sl.	5.6
4. S. T.	v. sl.	5.0, 6.0
5. H. A.	v. sl.	6.2, 5.6
6. B. W.	sl.	2.8, 4.0, 3.2, 3.81
7. K. G.	sl.	4.4, 4.6
8. E. H.	sl.	4.4, 4.6
9. B. A.	sl.	6.0, 6.0
10. S. J.	sl.	5.4
11. D. F.	+	3.7, 3.8, 3.0
12. P. A.	+	3.8, 4.3, 5.2, 5.1, 4.0, 4.6, 5.1
13. C. H.	+	4.2, 4.2
14. G. L.	+	4.3, 4.0, 4.3, 3.4, 4.0
15. R. T.	+	4.1, 4.0, 4.1
16. R. L.	++	3.9, 3.9
17. S. T.	++	3.1, 2.9, 2.9, 4.0
18. E. C.	++	4.0, 3.6, 3.7, 4.4, 3.2
19. A. A.	++	4.8
20. S. A.	++	4.2, 4.2, 3.7, 3.2, 3.6, 3.2, 3.8
21. W. R.	++	3.8, 3.9, 3.8
22. B. R.	+++	2.4, 2.4, 2.4

Haldane has shown that extreme and intolerable hyperpnœa develops in the healthy subject when the air in the bag contains 5.6 per cent. of CO_2 .⁵ Other observers have noted that some subjects reach a percentage of 6.2 CO_2 before the breaking point is reached. The figure is a variable one for normal subjects.

The patients who had very slight dyspnœa on exercise attained an average of 5.8 per cent. CO_2 . As the severity of the cases increased, the percentage of CO_2 tolerated decreased, and reached the low figure of 2.4.

Haldane,⁴ in experiments with a 50 litre cylinder of air, found that the factor of oxygen decrease played no part unless the initial percentage of oxygen in a mixture of oxygen and nitrogen was below 15. In none of the experiments was the initial percentage of CO_2 below this figure. When CO_2 was added it was only added to bring the initial amount up to 1 per cent. It may, therefore, be assumed that the results are consequent upon the accumulation of CO_2 and not upon oxygen want.

These patients show an intolerance to accumulating CO_2 .

SUMMARY.

The following conclusions apply to patients classed as suffering from "irritable heart."

1. The percentage of CO_2 in the alveolar air taken at rest is within the lower limits of normality or is decreased.

2. The reaction of the alveolar CO_2 pressure to exercise is similar to, and of the same order as, that found in the healthy subject. There is an increase during exercise followed by a decrease after the exercise is finished. Very mild exercise produces the same reaction in these patients as severe in healthy subjects.

3. The time during which the breath could be held is much less than in healthy subjects.

4. The percentage of CO_2 in inspired air which produces intolerable hyperpnœa is below normal, except in the very mild cases.

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MASSAGE OF THE FIBRILLATING VENTRICLES.*

· BY A. GOODMAN LEVY.

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THE earliest experiments in massage of the heart were performed by Schiff,^{1*} who maintained that its function was to establish an artificial circulation, and that it should be associated with a system of artificial respiration. With this view later observers are generally agreed.

Cardiac massage has been studied following three types of cardiac failure, induced by (1) asphyxia, (2) an excess of chloroform, (3) fibrillation from electrical excitation. Prus¹⁵ has demonstrated that rhythmic compression of the ventricles associated with perflation of the lungs is an efficient means of restoring an energetic ventricular beat following syncope from asphyxia and chloroform poisoning: the method was successful up to the maximum of one hour from the moment of syncope to the application of massage. These experiments were performed on dogs.

Prus further applied this method to 35 dogs in which syncope was due to ventricular fibrillation caused by the application of an electric discharge to the heart, and in this series he only succeeded in restoring a regular and energetic beat in five instances. In 29 of the remaining cases some degree of spontaneous beat was restored by persistent massage, but this did not become sufficiently active to sustain the circulation.

I have experimented on two dogs, in which ventricular fibrillation was induced by adrenalin under light chloroform anæsthesia, and the results accord with those of Prus. Prolonged massage failed to restore an active beat, but in one case a feeble ineffective ventricular beat was obtained after $11\frac{1}{2}$ minutes of massage.

Batelli¹ has been successful in restoring the beat of the fibrillating ventricles by the application of an alternating electric current of 240 volts, but this method does not appear to have a practical application.

It has been very generally held that ventricular fibrillation is a final and irrevocable catastrophe, but I am inclined to think this view has arisen from observations on dogs only. D'Halluin⁴ observes that there is a tendency to spontaneous recovery which varies in different animals: he says that fibrillation is most often found to be transitory in cats, but in the dog the fibrillating ventricles never recover their beat spontaneously.†

* A grant in aid of the research was made from the Graham Research Fund.

† This observation is not altogether correct, for Nobel and Rothberger¹³ publish an electrocardiographic record of transitory fibrillation in a dog.

Ventricular fibrillation in the cat's heart. In cats there is a conspicuous tendency towards spontaneous recovery from ventricular fibrillation, however produced. In a recent series of 20 experiments on cats there were five instances of recovery from fully established fibrillation produced by the chloroform-adrenalin reaction.

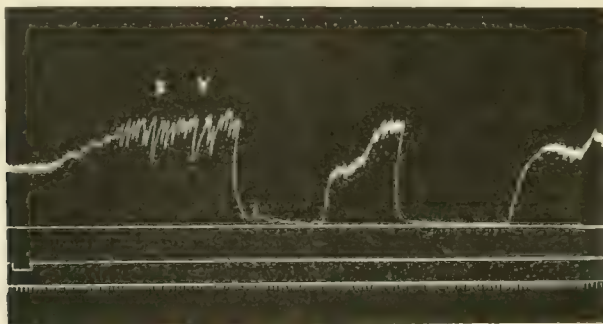


Fig. 1, $\times 2/3$. Hürtle manometer. Curve illustrating recovery from ventricular fibrillation. Cat under 0.8 per cent. chloroform. The signal marks denote the injection of 0.064 mgm. of adrenalin. For further description see text. Time marked in seconds.

Fig. 1 is a blood pressure curve from an experiment in which fibrillation was produced by the chloroform-adrenalin reaction, and shows complete syncope and recovery; the partial falls of blood pressure occurring in the preliminary stage of multiple tachycardia (marked thus, \times) are momentary phases of fibrillation, as I have demonstrated elsewhere ("Heart," 1912-13, IV, 326). The recurrent syncope is evidently due to the adrenalin still being active; the second recovery is permanent.

In the case of the adrenalin reaction I have never observed spontaneous recovery to occur after a longer interval than 1 minute from the moment of fibrillation, and rarely so long; that fibrillation may occasionally cease at a later period I have no doubt (see *infra*, page 178), but then the ventricles being completely bloodless and asphyxiated, are incapable of taking up a spontaneous beat.

When the cat's ventricles are caused to fibrillate by a short application of a faradic current recovery is more frequent than permanency of fibrillation; in this case recovery may occur after a rather longer interval, 1' 20" being the longest observed.

The tendency to recovery thus occurs during the active and visibly evident, or "first stage," of fibrillation, which continues for one minute, or thereabouts. Thereafter the ventricles dilate and in dilating lose their active movements, so that in about two minutes' time only faintly visible flickerings can be observed, and these often confined to the neighbourhood of the inter-ventricular septum. In this "second stage" of fibrillation

ample electrocardiographic oscillations are still registered,[§] but of rather slower rate, and these continue, but for how long has not been definitely ascertained.

If the ventricles in the second stage of fibrillation be compressed rhythmically in conjunction with perfusion of the lungs, they become less dilated and gradually resume their active and visible undulations: in the absence of artificial respiration the ventricles do not respond to compression, remaining in *statu quo*, or there may occur a very brief phase of active fibrillation* followed by a relapse to the second stage in spite of continued compression.

The foregoing observations point to the conclusion that the second stage of ventricular fibrillation is merely fibrillation in ventricles which have become asphyxiated through depletion of blood, and that the effect of rhythmic compression with artificial respiration is to remove this asphyxial condition. That such is the case is demonstrated by the bright red colour of the left auricular appendage, which can be permanently maintained by massage.

Active fibrillar movements are maintained so long as massage is continued, but not always in the same degree of intensity: this fluctuates from time to time, and, especially after prolonged massage, it assumes its maximum after a short cessation of massage.† Doubtless, ventricular fibrillation is comparable to the condition of auricular fibrillation in man, in whom the fibrillation continues indefinitely under the conditions of a sustained circulation.

In my view the function of cardiac massage is to restore the fibrillating ventricles to a condition, *i.e.*, the first stage of fibrillation, in which they can exert their natural tendency to recovery, and to maintain this condition until recovery actually takes place, which invariably occurs sooner or later. Massage does not provide a mechanical stimulus to recovery, which frequently occurs during a pause in the process (Fig. 2).

The conditions attending the recovery of the ventricular beat following massage. The transition from fibrillation to a regular beat is quite sudden: generally the beats are powerful and cause the blood pressure to mount up rapidly, as in the case of spontaneous recovery (Fig. 2). Occasionally, however, the blood pressure does not return to the same height as it assumed before fibrillation, and this is especially the case following a prolonged period of asphyxia of the ventricles, or after prolonged massage.

The resuscitated beats are regular, and appear to be sequential to the auricular beat: in the chloroformed animal the beats may soon relapse into the multiple tachycardial type if the recovery takes place very soon after

* No doubt because the blood has not become fully deoxygenated.

† Gunn & Martin (*Journ. Pharmacol. and exper. Therap.*, 1915, vii, 31) find that when massaging the depressed heart, the spontaneous beat develops more readily during a pause; similarly it is probable that prolonged manipulation of the fibrillating ventricles is deleterious to the resumption of active fibrillar movement.

the onset of fibrillation (Fig. 1), but they remain regular when recovery takes place later.

Sometimes the auricles assume a fibrillar state as well as the ventricles, but this is not permanent, and even when existing it is not a bar to recovery, for I have seen both auricles and ventricles pass simultaneously from fibrillation to the regular beat: I have likewise seen the auricles remain fibrillating and the ventricles pass from fibrillation to the characteristic response to auricular fibrillation. On a few occasions I have seen massage cause recovery without the intervening phase of active fibrillation: after a few compressions at first only a feeble ventricular beat is manifest, and this

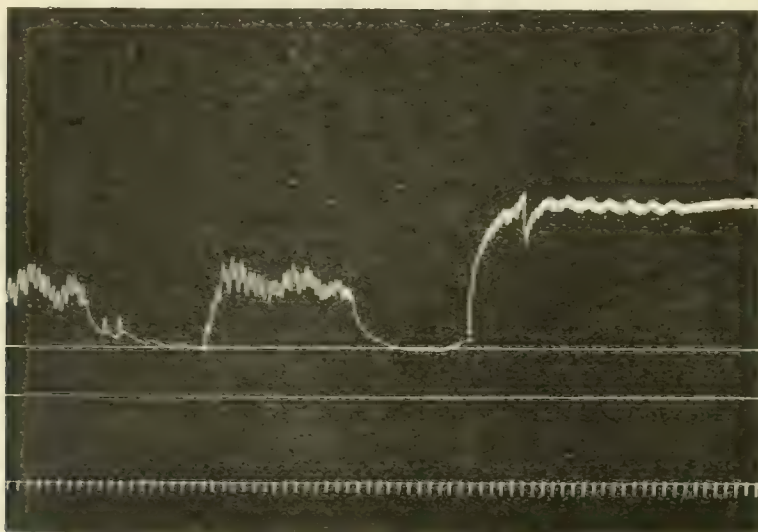


Fig. 2. Full size. Hürtle curve illustrating massage of the fibrillating ventricles followed by recovery. The massage is intermitted and recovery is shown occurring during a pause in massage. Artificial respiration. Time marked in seconds.

increases in strength somewhat rapidly as massage is continued, but in a different manner to the sudden bounce into activity usually observed. I believe that such cases are instances of fibrillation having already died out, and all that the ventricles require is oxygenated blood to enable them to take up a spontaneous beat: the manner of the appearance of the beats is in favour of this view.

Experimental methods and results.

The main factors of success are evidently mechanical: firstly, an efficient artificial respiration, and, secondly, an efficient artificial circulation. Care is required that the right ventricle is fully compressed in order to ensure that blood is forced through the lungs.

The rate of massage is relatively unimportant, but it should err on the side of rapidity, to ensure aeration of the blood, and to maintain a certain

degree of blood pressure, which appears advisable in order to fill the coronary arteries.

The value of intermittent massage is undoubted in such cases in which recovery does not occur within the first five minutes ; short pauses up to 45 seconds in duration then conduce to very active fibrillation with frequent transition to a spontaneous beat.

Two methods of causing fibrillation were employed—

- (1) The chloroform-adrenalin reaction ;*
- (2) Direct application of a faradic current to the heart muscle.

The first method was preferred, for fibrillation was obtained with less preparation of the animal, the chest being opened for purpose of massage at varying periods *following* apparent death. The details of the second method are as follows :—

The cat was anaesthetised with chloroform to the full surgical degree, following which artificial respiration was set up *via* the trachea. A cannula was introduced into a carotid artery for the registration of pressure curves by means of a Hürtle manometer : the manometer fluid was half-saturated sodium sulphate solution. The chest was opened by a left lateral incision and all bleeding points clamped. A small window was cut out of the pericardial sac, large enough to allow of direct application of the electrodes to the heart, but the main purpose of the window is for inspection of the heart, for the application of the electrodes externally to the pericardium is effective in producing fibrillation. The chloroform was reduced to 0.5 per cent. to allow the heart to beat vigorously previous to applying the current. This current was a faradic one, and the moment of fibrillation was reckoned to coincide with its application, for with a strong current the pressure falls to zero immediately, even though a true fibrillation may not be set up at once. The electrodes were removed after a few seconds, and if the ventricles were found to be not fibrillating they were applied again after an interval. Sometimes an application of 15 or more seconds was necessary to ensure permanent fibrillation. The carotid was clamped, following fibrillation, to prevent reflux of the cannula fluid during prolonged massage. Ten of the experiments were performed without taking a pressure curve, in order to exclude any influence of the soda sulphate solution, but with no difference in the results.

There is little difference between the methods in regard to the result, but the ventricular beat appears to be more frequently feeble on recovery when fibrillation is induced by the second method.

* This reaction is obtained as follows :—The cat is anaesthetised under a continuous stream of 2 per cent. chloroform vapour (increased in strength, if necessary) forced through the stem of funnel which fits loosely over the animal's head. When fully under, a superficial vein is exposed, and the connection of the manometer to a carotid artery effected. The percentage of chloroform is then reduced by gradual stages, until a fairly brisk corneal reflex has returned, and the vapour is under 1 per cent. At this stage the intravenous injection of 0.064 to 0.128 milligrammes of adrenalin generally results in fibrillation of the ventricles in from 10 to 20 seconds.

In the two earliest experiments the heart was massaged indirectly by a finger passed under the diaphragm, and the heart compressed against the chest wall, which was unopened. The chest was simultaneously compressed by the fingers of the other hand above the heart, in order to resist its tendency to slip away from the finger below. In the first experiment massage was successful in $2\frac{3}{4}$ minutes, but was unsuccessful in the second after $7\frac{1}{2}$ minutes. The chest was then opened and direct massage was rapidly successful. It was evident that compression was not very effectual when performed in this fashion, and in all later experiments the chest was opened and the heart compressed between the thumb and fingers applied outside the pericardial sac.

TABLE I.

Interval from moment of syncope to commencement of massage.	Duration of massage before ventricular beat returned.
2' 0'' { A	19' 30'' 0' 20''* 1' 0''
2' 0'' { F	0' 10''* 1' 0''* 9' 35'' 1' 0''* 4' 0'' 12' 30'' (a)
2' 30'' { A	2' 0''
2' 30'' { F	4' 30'' 9' 0''
3' 0'' { A	16' 0'' 5' 30'' 0' 20''*
3' 0'' { FF	2' 50''
4' 0'' F	3' 0''
5' 0'' F	0' 30''*
7' 0'' F	48' 0''
8' 0'' F	22' 25'' 6' 10''
9' 0'' F	23' 0''
10' 0'' A	3' 15''
11' 0'' { A	5' 40''
11' 0'' { F	4' 0''
12' 0'' A	8' 0''
13' 0'' F	4' 10''
15' 0'' { F	7' 0'' (a)
15' 0'' { A	24' 30''

A = Adrenalin reaction.

F = Faradic reaction.

In those experiments marked by asterisks the ventricles responded to massage without an intermediate stage of fibrillation. In two of these cases the ventricles appeared to be beating extremely feebly before massage was commenced.

The results of experiments on 29 cats have been summarised in Table I ; in every one of these experiments massage was successful in restoring the ventricular beat. The absolute efficacy of the method in cats is thus remarkable. In one case (*a*) the ventricular beat on recovery was feeble, and barely registered on the kymograph ; an intravenous injection of adrenalin raised the blood pressure, and further massage soon restored an entirely satisfactory circulation. In one other case (*b*) the ventricular beat was rapid and weak on recovery, and further massage for 6 minutes was required to make it satisfactory. In three other cases the blood pressure on recovery was rather low but fully efficient. Otherwise the circulatory conditions on recovery were entirely satisfactory.

Apart from the foregoing series of experiments, I have records of one experiment which may be regarded as a failure. In this instance a successful experiment had already been performed, and the heart caused to fibrillate a second time by a further injection of adrenalin under chloroform ; massage was begun 10 minutes after fibrillation, but there was a difficulty in emptying the heart and in obtaining a pressure curve during its performance. Only faint fibrillar movements were observed, and after 14 minutes of massage the pericardium was cut open in order to inspect the heart more closely ; in doing this the left ventricle was accidentally cut and the experiment terminated. A small clot was found in the entrance to the aorta, which was probably the cause of the failure.

The fortuitous duration of massage necessary before recovery occurs is obvious from the foregoing table. It is, I think, evident that given opportunities of sufficient duration the ventricles will sooner or later exert their natural tendency to recovery.

The longest interval allowed to elapse between the occurrence of syncope and the commencement of massage was 15 minutes, as a longer interval did not appear to bear considerations of practical importance, as will appear later in this paper. The average time of recovery, after an interval of 11-15 minutes was 8' 40'' which does not greatly exceed the average following the interval 1-5 minutes, viz., 5' 8''. The average following the 6-10 minute interval was much higher, viz., 20' 34''.

In the first series with a 1-5 minutes' interval, there were 6 cases of rapid recovery, fibrillation having died out, and these are responsible for the lower average of time of recovery in this period ; otherwise there is little difference between the first and last 5 minute period. Four of these early recoveries occurred after an interval of 2 minutes only.

A review of the experimental records of the whole series shows that the earliest recoveries were generally obtained with an ample artificial ventilation of the lungs, and an efficient compression of both right and left ventricles.

The employment of drugs in combination with massage. In a recent paper McWilliam¹¹ states he was successful in restoring the fibrillating heart by the use of certain drugs, used either singly or in combination, followed by massage. The drugs he injected either into the heart muscle, the ventricular cavities, or the jugular vein.

I have performed experiments with a number of these drugs, but the results obtained have not been so far superior to those obtained by massage alone as to commend their routine employment. It is intended to extend this investigation.

Massage of the heart overdosed with chloroform. It appeared desirable to obtain records of the effect of massage on the overdosed heart to compare with that on the fibrillating heart, and a number of resuscitated animals were employed for this purpose; a few experiments were performed on fresh animals with similar results.

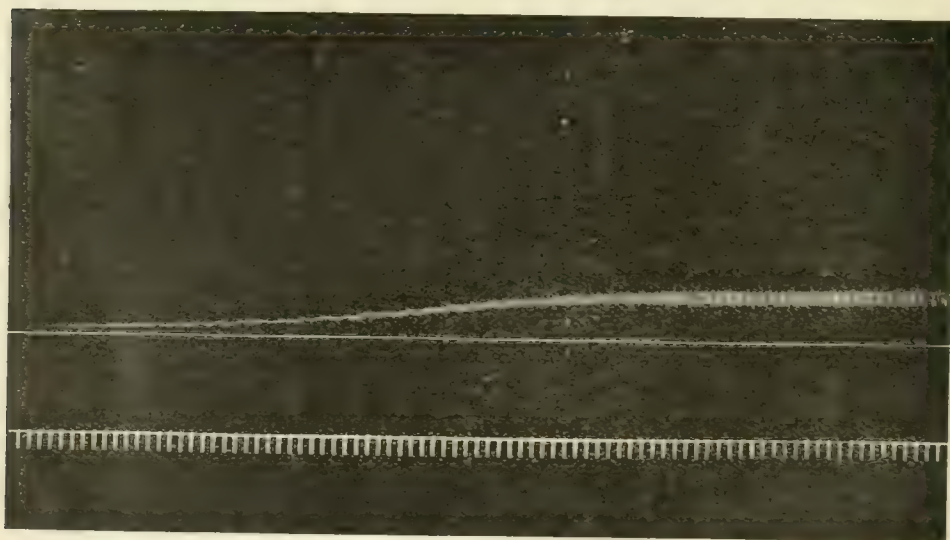


Fig. 3. Full size. Hürtle curve illustrating recovery of the heart from an overdose of chloroform following massage under artificial respiration. Time in seconds.

Prus¹⁵ overdosed his dogs by allowing them to inhale chloroform until the respiration and heart's action had ceased, and then deferred massage for periods up to one hour. The action of cats' ventricles is not entirely suppressed by the action of chloroform alone administered in this way, and I therefore in several cases completed the intoxication of the heart by massage of the ventricles whilst maintaining artificial respiration with an atmosphere containing a high percentage of chloroform; in this way the action of the ventricles was entirely suppressed by chloroform poisoning alone, apart from the asphyxia. These hearts were therefore far more deeply affected by chloroform than would be the case of an ordinary case of cardiac

failure from overdose. Seventeen experiments were performed and all with success : there was no difficulty in restoring the overdosed heart by massage combined with artificial ventilation of the lungs with air. The best results were those in which care was taken to fully compress the right ventricle : it is sufficient to record my last three experiments in illustration of the results obtained (see Table II).

TABLE II.

In each experiment the animal was fully anaesthetised with chloroform before the overdose was administered.

No.	Time.	Procedure.	Remarks.
Cat 1	0' 0"	3.5% CHCl ₃ by artificial respiration begun.	Ventricular beat extinguished.
	3' 0"	Massage under 3.5% CHCl ₃ .	
	5' 30"	Massage ceased.	
	8' 0"	Massage recommenced under A.R. with air only.	
	8' 20"	Massage ceased.	Very faint ventricular beat.
	9' 0"	Massage resumed.	Beat stronger, improves with out further massage.
	9' 10"	Massage ceased.	
	10' 0"		Beat nearly normal.
Cat 2	0' 0"	4% chloroform by artificial respiration.	Ventricular beat very feeble.
	2' 0"	Massage commenced under 4% CHCl ₃ .	
	4' 0"		
	5' 0"	Massage ceased.	Ventricular beat entirely extinguished.
	6' 0"	Massage recommenced under A.R. with air only.	L. appendix beat almost extinguished.
	7' 30"		L. aur. appendix beats rapidly, ventricles respond feebly.
	8' 0"		Ventricular beat stronger.
	8' 30"	Massage ceased.	Beat improves gradually.
	10' 0"		Strong heart beat.
Cat 3	0' 0"	3.5% chloroform by artificial respiration begun.	Ventricular beat feeble.
	2' 30"		
	3' 15"	Massage under 3.5% CHCl ₃ .	
	6' 0"	Massage discontinued.	Ventricular beat extinguished.
			L.A. faint flickering beat.
			R.A. beats well.
	11' 0"		L.A. motionless. R.A. beats feebly.
	11' 15"	Massage under A.R. with air only.	L.A. and V. beat returning.
	12' 0"		Heart beats moderately strongly.
	12' 45"		
	13' 0"	Massage ceased.	Beat gradually improves.
	13' 45"		Beat almost normally strong.
	15' 30"		

The mode of recovery of the heart from overdose is characteristic. At first a very feeble beat appears which requires further massage to improve it. This beat when strong enough to register on the kymograph gradually improves without further massage. An illustration of such a recovery is given in Fig. 3, and this curve is in striking contrast to that of the typical recovery from fibrillation (Fig. 2). This form of recovery is consistent with the view that the massage effects an artificial circulation and the heart regains its action progressively as the blood throws out its chloroform.

CLINICAL CONSIDERATIONS.

The clinical importance of these experiments arises more especially out of the causal relation of ventricular fibrillation to chloroform syncope, a matter which has been fully dealt with in former publications."

The question arises how far do these experiments on fibrillating ventricles apply to the heart of man. Does this react like the heart of the dog or that of the cat?

The answer to this question is evidently involved in a further question: Do the fibrillating ventricles in man exhibit a tendency to spontaneous recovery as in the cat? If so, then it should be possible to foster this tendency to recover by massage.

Definite evidence of spontaneous recovery in man is afforded by Robinson and Bredeck¹⁶ in the form of an electrocardiographic record of a syncopal attack with recovery in a woman, showing ventricular fibrillation. The length of attack is stated to have been about four minutes, but it seems extraordinary that the ventricles should be able to take up their beat after such a long period of asphyxia; possibly in the absence of an accurate record the period is exaggerated. There were several similar attacks, presumed to be of the same nature.

Hoffman⁶ has also recorded a momentary period of fibrillation following a tachycardial attack; but his interpretation of the tracing has been called in question.

Apart from these records, there is abundant evidence of spontaneous recovery in clinical reports of chloroform syncope in man. Seymour Jones⁷ records 100 cases of operation for deflected septum of the nose in patients lightly anaesthetised with a chloroform and ether mixture, a submucous injection of adrenalin being given to secure a bloodless field. It is evident that the characteristic small rapid and irregular pulse frequently followed the injection, and there were three cases of complete cardiac syncope with sudden intense pallor, extreme dilatation of the pupils, followed by cessation of respiration. All these three cases of syncope, which can safely be attributed to ventricular fibrillation, recovered spontaneously after a brief period, the length of which was not definitely recorded.

About the same time Blumfeld² and others recorded similar experiences as the result of the injection of adrenalin under chloroform. That death not infrequently results in this way was pointed out by myself shortly afterwards,³⁰ and I find I made the following comment upon the facts at the time. "It is evident from these clinical cases that the human heart has a greater capacity for recovery (*i.e.*, from ventricular fibrillation) than has the feline heart."

Further, it is no uncommon event for recovery to follow syncope of sudden cardiac origin in the course of the administration of chloroform alone. One anæsthetist of my acquaintance informs me that he has frequently observed the characteristic sudden pallor followed by deep sighing respirations, but that death has been the exception, and a number of examples may be found in the report of the Anæsthetics Committee of the British Medical Association, 1900 (*e.g.*, Nos. 607, 608, 614, 618, etc.).

Inasmuch then, as the human heart has evidently an undoubted tendency to spontaneous recovery from ventricular fibrillation, we should expect massage to be almost uniformly successful in such a condition if properly performed.

Conditions appertaining to successful massage in the human subject.

(1) It is evident that the duration of the interval between the moment of syncope and the commencement of massage is not closely connected, so far as recovery of the heart's action is concerned. Certainly I obtained several rapidly successful results when applying massage not later than the first two minutes, but as at least this period should be allowed to elapse before undertaking any operative procedure, in order to see if spontaneous recovery supervenes, it is not generally allowable to commence massage so soon. There is, however, a distinct limitation to the length of this interval, governed by the fact that the nervous centres are impaired by prolonged asphyxia.

Stewart, Guthrie, Pike and Burns³¹ concluded from the ligation of the cerebral blood vessels in animals that 16½ minutes was the longest time that the brain could be denuded of blood without permanent injury. A. W. Russell³² judges from a review of these experiments coupled with clinical experience and the reports of cases of recovery after massage that "we are probably safe in assuming that about five minutes' loss of its circulation is the outside limit of time that the human brain can withstand and recover completely." He cites the reported cases of completely successful massage in support of his view, in which the interval of complete anæmia was under five minutes, with the exception of one case, in which it was estimated at 7 to 8 minutes.

He also cites a case reported by Green⁵ to show the effect of prolonged anæmia. This, briefly, is as follows :—

M. 9 years. Operation for umbilical hernia. Anæsthetic—chloroform. Movement of patient followed by collapse.

12.30 p.m. Heart stopped.

12.55 Massage, sub-diaphragmatic, and artificial respiration.

12.58 A slight fluttering of heart felt, followed in a few seconds by a vigorous beat. Sensibility never returned. Tonic spasm and rigidity came on and the patient died in 20 hours.

However, more recently Mollison¹² has published a case in which the interval was certainly not less than 13 minutes and probably was 15 minutes. In spite of severe nervous symptoms, this patient ultimately recovered.

Brief details are as follows :—

M. 6 years. Operation for tonsils and adenoids. Anæsthetic—chloroform and ether mixture.

1.5 p.m. Heart stopped.

1.20 (probably), massage, sub-diaphragmatic, begun.

1.24 (probably), heart commenced to beat.

Artificial respiration was not performed, but spontaneous respiratory movements continued intermittently.

The patient was unconscious more or less for seven days, during which he presented features of severe cerebral irritation, with rigidity, tetany, screaming and incontinence of urine and feces. He was much improved in 14 days and eventually recovered completely.

A recovery such as this is exceptional, but it is evident that the interval of 5 minutes assigned by Russell may be somewhat exceeded with a good prospect of success, and that the case need not be regarded as absolutely hopeless even after an interval so long as 13 minutes.

Another matter to be considered in regard to the latitude of time allowable before commencing massage is that of intra-cardiac clotting. On this point we have the evidence of Pike, Guthrie and Stewart,¹⁴ who found the earliest evidence of clot in the right ventricle 36 minutes after death, whilst Sollman found clots in the heart in two animals out of three in 20 minutes after death. In both series the moment of clotting is subsequent to the latest time allowable before commencing massage as already determines in regard to the condition of the nerve centres, so that clotting need not be taken into consideration as a matter of practical importance.

(2) The technique of obtaining proper access to the heart and compressing it is evidently of great importance. It is essential the heart should be thoroughly grasped and the contents of both ventricles fully expelled, in order to irrigate the capillaries of the lungs and the heart.

In a number of recorded cases of the practice of massage the heart has been exposed by resecting the costal cartilages and opening the pericardium, but this method has been attended with a single success only. It is a somewhat severe operation, and the delay incurred on this account is a serious disadvantage.

The subdiaphragmatic mode of approach has nearly all the recorded successes to its credit, but I believe likewise a vast number of failures. It

can rarely be entirely satisfactory. Bost and Neve² say that compression of the heart in this way is possible though difficult in the child, but in the adult it could rarely be effected, as only the cardiac apex can be reached, and this tends to slip upwards. Further, that it is possible to obtain access to the interior of the pericardium by an incision into the base of it from below the diaphragm. The left lobe of the liver and the stomach are, however, liable to get in the way: there is a risk of injuring the musculophrenic artery, and there is considerable difficulty in inserting sutures in the diaphragm and finally close the incision. Bost and Neve therefore have practised another method, briefly as follows: An abdominal incision is made and through this the attachments of the diaphragm to the left costal margin are cut for two inches. The right hand is inserted into the left pleural cavity and the heart grasped outside the pericardium. During massage the parts are pressed around the right wrist to prevent air entering the pleural cavity. This method appears to me to satisfy most nearly the required conditions.

(3) The performance of artificial respiration. Success as a result of cardiac massage is almost out of the question without artificial respiration: there is in fact abundant evidence that its performance is generally neglected.

In animals, shortly after massage is begun in conjunction with perflation of the lungs, spasmodic gasps of an asphyxial character occur at irregular intervals, which disappear as soon as the heart recovers its spontaneous beat, but I do not think I have ever seen any attempt at regular respiratory efforts whilst massage is being performed. In man, as in cats, as judged from clinical records, there is frequently a strong tendency to the persistence of respiration after cardiac failure, and no doubt cardiac massage will assist to maintain them if performed soon enough. Such effects can, however, only be fitful and quite inefficient for the purpose in view as a general rule, although it would appear that recovery by massage has occurred in a few instances without artificial respiratory assistance.

Probably artificial respiration performed by Sylvester's method is sufficient when there is no opening into the pleural cavity, provided it does not interfere with the proper performance of massage. In the case of an operation such as suggested by Bost and Neve, undoubtedly perflation of the lungs would be much more safe and effective.

Intubation of the larynx can be easily performed on an individual in the flaccid condition following cardiac syncope, when the initial asphyxial spasm has passed off, and the lungs can be readily blown up through the tube by the lung power of another person. Perflation of the lungs by means of the apparatus employed for the intratracheal method of administering an anæsthetic should likewise be effective. Even mouth to mouth perflations may be practised in an emergency, but one or other method must be employed in an efficient manner if cardiac massage is to be made a practical success.

(4) *Posture.* Partial inversion of the patient is no doubt beneficial by keeping the cerebral centres bathed with blood, and also by assisting the filling of the heart. There is no experimental evidence that it assists recovery in any other way.

Massage of the heart overdosed by chloroform. When syncope occurs from an overdose of chloroform, the performance of artificial respiration alone when promptly performed is sufficient to restore the patient, but it is no doubt insufficient when deferred too long. In such a rare case cardiac massage might be necessary, and should easily effect a remedy. The technique would be precisely similar to that relating to the fibrillating ventricles.

CONCLUSIONS.

(1) In the cat the fibrillating ventricles can be restored to a regular beat by means of rhythmic compression combined with artificial respiration.

(2) The explanation of the action of massage in such circumstances is the production of an artificial circulation of oxygenated blood through the coronary arteries. In this way the ventricles are made to resume active fibrillation (or the "first stage" of fibrillation) in which they exert a natural tendency to revert to a rhythmic beat. The ventricles cannot resume their beat in the "second stage" of fibrillation, which is the result of the asphyxia of the fibrillating ventricles.

(3) The human heart possesses a natural tendency to recovery from fibrillation, and should therefore be susceptible to treatment by cardiac massage.

(4) The successful performance of massage depends upon the efficient compression of both ventricles and an efficient system of artificial respiration. Continued rhythmic compression should be tried at first, and if this is not successful in the first ten minutes, it should be intermitted by short periods up to 45" duration.

(5) In the interest of the nerve centres massage should be commenced not later than five minutes from the moment of cardiac failure.

(6) Two minutes is probably the outside limit of time which should be allowed for the chance of spontaneous recovery in man, after which the necessary operative procedures should be undertaken.

(7) There is good reason to believe that cardiac massage, if carried out in an efficient and persistent manner, should prove an effective means of restoration from apparent death due to chloroform syncope, whether this be the result of ventricular fibrillation or neglected overdoses.

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OBSERVATIONS UPON FLUTTER AND FIBRILLATION.

PART II.—THE NATURE OF AURICULAR FLUTTER.

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Preliminary note.

IN the observations which are described in this paper and in those which will follow it, the auricle has frequently been examined by means of direct leads. Before proceeding to a description of the main experiments, the method of leading may be described in more detail, and attention may be called to the information obtained by means of it.

The electrodes usually placed on the auricular surface consisted of a pair of small glass tubes, plugged with kaolin and containing a saturated solution of copper sulphate: in this solution the copper wires connecting to the string galvanometer were immersed. The centres of the two contacts were 8 millimetres apart. For descriptive purposes it will be convenient to name them. We name the one contact the Z contact (black in the figures) and the other the C contact (white in the figures). The Z contact is that which is connected to the same end of the recording fibre as is the right-arm electrode in human electrocardiography, as it is now generally practised. If this Z contact is connected to the zinc element, and the C contact to the copper element, of a copper-zinc couple, the resultant deflection is upright in our curves. An upright deflection of this kind indicates relative negativity of contact Z. If an upright deflection occurs as a result of muscular activity, that deflection indicates primary activity of the muscular tissue lying beneath the Z contact. This is well known as a result of long experience, and may be illustrated by means of a simple experiment. Fig. 1 shows a pair of contacts lying in the middle of the exposed surface of the right auricle and connected to the galvanometer. If a wave of excitation is started by stimulation in the neighbourhood of A, it will travel to the muscle under the contacts, and will pass under them in the line of the contacts as indicated in the diagram. The excitation wave reaches the muscle lying under contact

Z first of all, and this contact becomes negative to *C*: a sharp and upright deflection of the string results* (see Fig. 20, *A*). If the stimulus is applied at *B*, a deflection of similar magnitude is seen, but its direction is reversed: it is directed downwards (see Fig. 20, *B*). If the stimulus is applied at *C*,

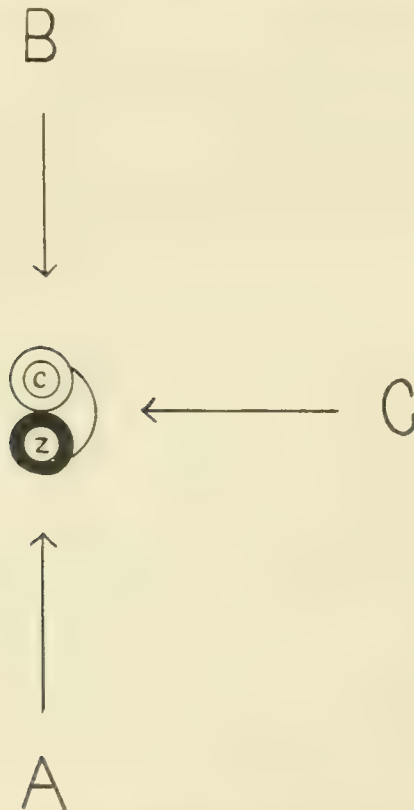


Fig. 1. A diagram illustrating the method of examining the auricle by means of a direct lead. Two non-polarisable contacts *Z* and *C* are placed on the auricle and joined to the galvanometer. The *Z* and *C* contacts are so named because, if *Z* is joined to the zinc and *C* to the copper element of a copper-zinc couple, at the moment of completing the circuit an upright deflection is shown by the galvanometer. The same deflection is yielded when the muscle beneath *Z* becomes active, while that beneath *C* still remains inactive. The muscle of the auricle is stimulated at *A*, *B* and *C* respectively: the corresponding excitation waves travel along the lines indicated and produce the curves shown in Fig. 20.

a point lying at right angles to the line of the contacts, the deflection produced is of small magnitude, and frequently there are several of them (see Fig. 20, *C*).

* The deflection which actually comes first is a little downward deflection. This, as has been shown, is not due to the arrival of the excitation wave at the contacts, but to its movement through muscle before the contacts are reached. It is therefore spoken of as an *ectronic* deflection, as opposed to the chief deflection, which is termed the *intrinsic* deflection.⁴

In using paired contacts to examine the auricular surface while the heart is beating in response to inherent impulses, both the magnitude and the direction of the deflections are closely examined. The appearance of a prominent deflection, when the contacts are revolved into a particular plane as they lie upon the muscle, indicates that the line travelled by the excitation wave, which is under examination, is being reached; the direction of this deflection tells us whether the excitation wave is travelling up or down the line of the contacts. Thus, in leading from the auricle in the fashion described, evidence is obtained of the general direction in which the excitation wave is travelling. If the curve taken from this direct lead is inscribed simultaneously with a curve from a standard lead, for which purpose lead *II* (from the right fore-limb and the left hind-limb) is used, the arrival of the excitation wave at the point of auricular muscle examined may be accurately timed. It is timed relative to *P*, the auricular wave in lead *II*. Maintaining the standard lead and moving the direct lead over the surface of the auricle, the times of arrival at all surface points may be ascertained and related to each other. Thus, the course of the excitation wave may be mapped out, and the course, thus indicated, may be compared with that suggested by the direction of the deflections in the direct lead.

This method has been employed extensively, and successfully, in studying the course of the normal excitation wave throughout the auricle; the results have been embodied in a recent paper.⁴ The present paper describes similar observations upon the auricle beating, not normally, but in a manner identical with what is known clinically as flutter.

Method for inducing flutter.

In experimenting upon dogs, which, being of sufficient size, are the only suitable animals available for our purpose, we have anæsthetised them fully by means of morphia, paraldehyde and ether.* The auricle is exposed by splitting the sternum, by tying back the side walls of the chest, and by opening and stitching the pericardium to the chest wall; the vagi in all experiments have remained intact.

Flutter has been induced either by weak faradic† stimulation of the auricle, or by rhythmic stimulation rising in rate until a critical point is reached. This critical point usually comes when the rate of stimulation rises to between about 350 and 500 per minute. Faradic stimulation is a less certain means of inducing continuous flutter than is rhythmic stimulation at the rates named; but we have found no certain method.

By means of faradic or rapid rhythmic stimulation, every dog's auricle may be induced to yield an after-effect when stimulation is withdrawn;

* A statement which applies to all the experiments reported in this series of articles.

† Which is essentially rapid and rhythmic stimulation at a very high rate (2400-3000 stimuli per minute).

and these after-effects consist of flutter, of fibrillation, or of a condition which seems intermediate between the two. Some auricles are prone to fibrillate, and in these, with very few exceptions, we have been unable to induce pure flutter by any means: other auricles and these are less frequent, are prone to flutter, and often in these flutter may be obtained repeatedly.

A chief difficulty is in obtaining flutter, or fibrillation when it is desired, of sufficient duration. The observations are to be made during the after-effect, when stimulation has been withdrawn. In mapping out the auricle, during the progress of a flutter after-effect, an after-effect of considerable duration is essential. An after-effect lasting 20, 30 or more minutes is usually necessary. We know of no sure method by which these long after-effects are to be obtained, and this lack of knowledge adds much to the difficulty of the work. It is impossible to say at the moment stimulation is withdrawn how long the after-effect will last: it is even impossible to say with certainty that an after-effect will be obtained. On many occasions there will be no after-effect, on very many other occasions there will be a short after-effect, lasting a few seconds or perhaps a minute: on rare occasions an after-effect of many minutes' duration, very rarely of 30 or 60 minutes' duration, will be seen. It is upon these long after-effects that our observations have been undertaken. It will be apparent that there are many disappointments: some auricles will be stimulated repeatedly, and only fleeting after-effects will be obtained: in others longer after-effects will be obtained, but these will cease spontaneously before the mapping out is complete: in yet others the after-effect will be of sufficient duration, but more often than not it will consist of fibrillation or impure flutter, to which the method of mapping out is unsuited.

The duration of the after-effect, in our experience, is influenced neither by the strength of the induction shocks nor by the length of stimulation; but we believe that flutter is more easily induced, in auricles which are prone to show it, by stimulating the region of the tania terminalis, especially near the inferior cava, and that it is more likely to endure when this region of the auricle is chosen: it is difficult to be sure of this, however, because, wherever the stimuli are applied, the nature of the after-effect, and especially its duration, is uncertain. It is true that in any given animal the auricle seems prone to take up a certain abnormal rhythm, a subject to which we shall return in the next article of this series: it is also true, as we shall see, that two or more after-effects of short or of long duration may be obtained, which prove to be identical with each other or almost so. But this is not a fixed rule: consequently, in mapping out the course of the excitation wave, it is not safe to combine the results obtained during separate after-effects. The whole of the mapping out must be accomplished during the progress of a single after-effect: it is for this reason that the long after-effects are so much to be desired. There is a further consideration. The after-effect must present constancy throughout its course: the rate of beating must be constant, or must vary only in trifling degree, otherwise separate readings

are not comparable; there must be some guarantee that the mechanism does not alter. To ensure that the last condition has been fulfilled, the form of the auricular complexes in the limb-lead is a most useful guide: for if this shows no material change of form throughout the after-effect we may be certain that there has been no material change in the mechanism. The rate of beating is equally helpful, for if this is constant throughout, the mechanism remains constant.* It time permits, readings from the surface are repeated as an extra safeguard, though this can rarely be accomplished at all completely.

It will be clear from what we have said that the induction of flutter, sufficiently stable to last and to maintain itself as a constant mechanism, is not an easy matter in the present state of our knowledge and that the experiments are very time-robbing and often disappointing. By persistence, however, sufficient observations can be collected. The difficulties of the work form one reason why, in the case of successful experiments, we describe these in somewhat minute detail: for the possibility of their repetition on a large scale seems remote.

OBSERVATION 1. (*Dog JP.*)

The auricle of a dog of 13.5 kilos was exposed in the manner stated. By means of a special clamp, already described in this Journal, the auriculo-ventricular bundle was crushed and permanent and complete heart-block was produced.† The object of this procedure was to render the auricular electrocardiogram more distinct, for otherwise, when the auricle is in a state of flutter or fibrillation, the auricular portion of the curve is apt to be confused by the complexes of the rapidly beating ventricle.

First after-effect.

The auricular appendix was repeatedly stimulated by means of a weak faradic current. For the most part short after-effects were obtained subsequent to the end of stimulation. On one occasion, however, an after-effect lasting six minutes was obtained, and it is with this that we are at first concerned. During these six minutes the auricle beat regularly at a rate of between 376 and 392 per minute. Examples of the curve from lead *II* (taken from the right fore-limb and left hind-limb) are to be seen in Figs. 21 and 22. The auricular complex consists of a steep upstroke, which is followed by a steep descent of somewhat greater extent: the curve

* As Rothberger and Winterberg¹ have pointed out, longer after-effects are produced if the animal is injected with physostigmin or muscarin, before the auricle is stimulated. We have used this method, and it is true that long after-effects are obtained, but the method is open to the fatal objection that as the effects of the drug quickly wear off, the rate of beating falls and the mechanism changes. In attempting to map out the course of the excitation wave, this method of prolonging the after-effect proves valueless.

† The right division of the bundle was also damaged permanently by this clamping.

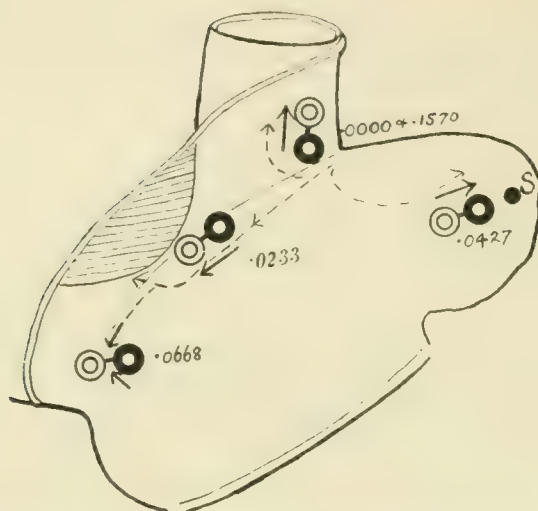


Fig. 2. Dog J.P. (Records 9-12.) A diagram of the ventral and right surface of the right auricle, showing the direct leads which were used during the first short period of flutter, which lasted 6 minutes. The Z contacts are drawn black and the C contacts white. The short unbroken arrows indicate the direction pursued by the excitation wave, as indicated by the direction of the deflections in the corresponding direct leads. The long broken arrows represent the course of the excitation wave as a whole, as judged from the direction of the deflections and the readings obtained from the several points examined. These readings are marked on the figure in decimal points of a second and are reduced to a new common zero. (See Fig. 4 and Table II.) S is the point of stimulation from which the flutter was induced; it lies on the tip of the right appendix.

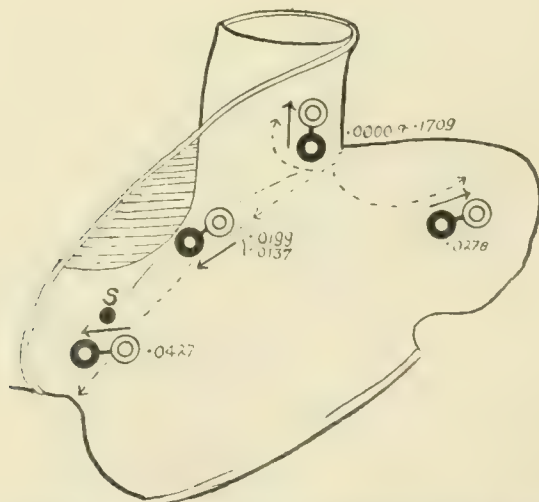


Fig. 3. Dog J.P. (Records 15-18.) A diagram constructed in the same fashion as the last. It sums up the first series of observations made during the second period of flutter; the flutter lasted 20 minutes. S, the point stimulated to induce flutter, lay over the inferior vena cava.

then rises gradually to its original level. There is no real iso-electric period. In some curves, but not in all, the summit is distinctly divided (see Fig. 23). The auricular complexes are contiguous; usually four complete complexes stand in each ventricular diastole and two coincide with and are more or less concealed by the ventricular systole. The auricular complexes are regular.*

During this short period of flutter four direct leads were used, and a curve from each of these four leads was taken simultaneously with a curve from lead *II*, the latter being used as a standard for measurement and to check the constancy of the mechanism which prevailed.

S. A. N.† lead (Record 9‡). The *Z* contact was placed over the supposed position of the head of the sino-auricular node, immediately on the vein side of the upper end of the sulcus terminalis; the *C* contact lay 8 millimetres above it on the vein. The corresponding curve is shown in Fig 21. The curve from the direct lead consists of a series of steep upward deflections of uniform shape and amplitude, arranged in rhythmic series. The degree of regularity with which the deflections succeed each other is shown by fine measurement of the curves. The lengths of successive intervals between adjacent deflections in this plate and the remainder to be described is given in Table 1. The average length of the intervals is 0.1529 of a second, and the greatest divergences from this average are +0.0026 and -0.0049 of a second.

The direction of the steep deflection shows that the excitation wave was travelling up the superior cava, against the direction of the blood stream. This is indicated in Fig. 2 by the unbroken arrow drawn immediately to the left of the contacts.

The readings, in seconds, given in this diagram will be explained presently; the dotted lines and arrows indicate the general course of the excitation wave.

The steep upstroke of the direct lead (intrinsic deflection)§ preceded the summit of the auricular complex in lead *II* by an average interval of 0.0482 of a second.

Mid-caval lead (Record 12). The *Z* contact was placed above and the *C* contact below, the pair being in the line of and upon the tænia between superior and inferior cava. The deflections were prominent and regular in incidence, amplitude and direction.|| They were upright, indicating the passage of the excitation wave down the tænia. The steep upstroke preceded the summit of the auricular complex in lead *II* by an average interval of 0.0249 of a second.

* The degree of regularity may be gauged from Fig. 21 and from the measurements of Table I. and other measurements to be described presently. It is of a very high order.

† *S. A. N.* is used as an abbreviation of sino-auricular node.

‡ This number is the number of the plate used in the experiment.

§ See footnote on page 192.

|| This plate was badly fogged, and although legible for measurement is not fit for reproduction.

Right appendix lead (Record 10). The contacts were placed a little distance from the tip of the appendix and in the line of the appendix, the Z contact being nearest the actual tip, where the stimuli originally provoking the flutter were applied (see *S* in diagram). The deflections were prominent

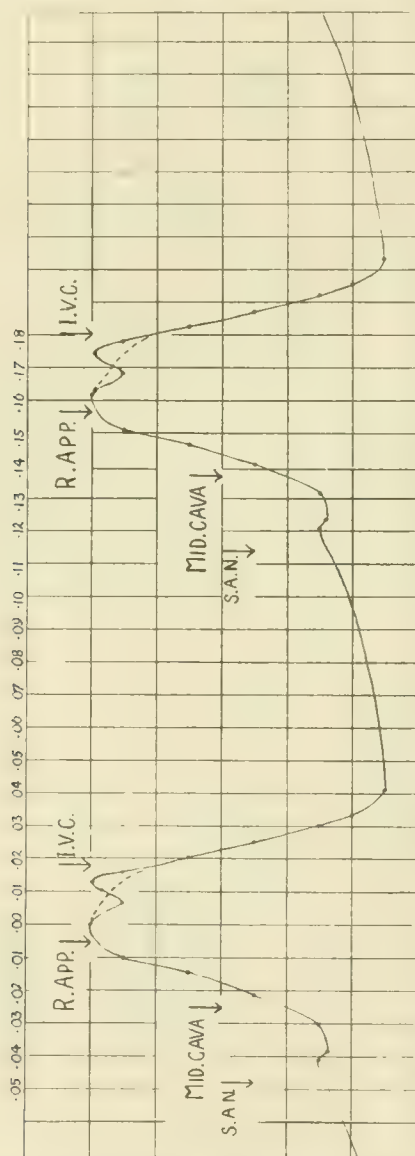


Fig. 4. *Def. I/P.* (Records 9-12.) A chart showing the time relations of the auricular complexes in lead *I/I* to the readings from four points on the surface of the auricle, during the first period of flutter. The actual leads are charted in Fig. 2.

The auricular curve in lead *I/I* has been plotted from a measured cycle of record 10. The arrows represent the times at which the excitation wave appeared, relative to the auricular complex, at the *S. I. N.*, mid-caval, right appendix and inferior vena caval regions. The measurements actually related these times to the first summit of the auricular complex. In Fig. 2, the new zero used in expressing the readings, is the arrow which marks the appearance of the wave at the *S. I. N.* region in the present chart.

During the progress of this first after-effect, the form of the auricular complex changed a little at its summit. In the early records the auricular summit was unnotched, as indicated by the dotted line.

The vertical lines mark hundredths of a second. The horizontal lines represent tenths of a millivolt.

and regular in incidence, amplitude and direction (Fig. 22). They were downwardly directed, indicating the passage of the excitation wave up the appendix toward its tip. The steep downstroke preceded the summit of the auricular complex by an average interval of 0.0055 of a second.

Inferior caval lead (Record 11). The contacts were placed in the line of the inferior vena cava, the *C* contact being closest to the actual vein. The deflections were regular in incidence, amplitude and direction. They were relatively small and there were several phases, but the chief deflection was upright* indicating that the excitation wave encountered the *Z* contact first: it did not pass in the immediate line of the contacts, but somewhat obliquely to them, as indicated by the alternative arrows in Fig. 2. The upstroke followed the summit of the auricular complex by an average interval of 0.0186 of a second.

Fig. 4 is a chart constructed from fine measurement of the curves. Auricular complexes from lead *II* are charted and arrows are placed above these so as to show the times in seconds, relative to the auricular complexes, at which deflections of the direct auricular leads appeared. The chart relates the arrival of the excitation wave at all the contact points to the electrocardiogram taken by means of lead *II*, and to each other. In the chart two complete complexes (of lead *II*) are charted. Actually the curves of both were charted from measurements of one: this method introduces no material error, because the auricular complexes were of equal length within a very small fraction of a second, and the times at which successive deflections in a direct lead appeared relative to the summit of the corresponding auricular complex varied by no more than negligible quantities. The charted values are each the average of four measured intervals.†

The readings of the deflections in the several direct leads, relative to the summit of the auricular complex are also given in the fourth column of Table II (1st after-effect).

In the fifth column of the same table, and for greater clearness, these readings are reduced to a new zero, namely, the time at which the excitation wave appeared at the contacts placed on the *S.A.N.* region. These readings of the fifth column are the ones used in Fig. 2.

During the period of flutter the excitation wave moved in the directions shown by the short unbroken arrows of our diagram: this is judged from the direction of the deflections in the direct leads. The times at which the deflections appeared relative to each other fully confirms this conclusion. The excitation wave moved down the tænia, up the superior vena cava and up the auricular appendix to its tip, as indicated by the broken lines and arrows.

It should be remarked that this movement through the appendix is contrary to that which would have prevailed during the period of stimulation. For the flutter was started by stimulating the appendix, the waves of excitation coursing during stimulation away from the point stimulated and therefore down the appendix.

* This curve is used in Part IV, Fig. 11.

† The maximal variations from the average for the four plates amounted to:—Record 9, 0.0049 and -0.0039 of a second; record 10, +0.0016 and -0.0021; for record 11, 0.0025 and -0.0015; record 12, +0.0021 and -0.0030.

Second after-effect.

The first period of flutter ended abruptly and without apparent reason. The faradic current was reapplied to the auricle; it was applied, not to the appendix, but to the inferior cava. Several brief after-effects were obtained, and finally the auricle passed again into flutter as a result of stimulation, and this period of flutter lasted 20 minutes.

During this period the observations of the first stage were repeated, so that the flutter of the first and second main after-effects might be compared. The same direct leads were used, though the arrangement of the *Z* and *C* contacts was not always the same as in the first after-effect. The actual arrangement of the contacts is shown in Fig. 3.

In experiments of this kind the curves are taken as quickly as possible, the flutter being usually of short duration. It is not always possible to be sure, therefore, that the pair of contacts is placed in exactly the same position as in previous observations. If time were less precious the auricle would always be sketched and small landmarks used to facilitate accurate replacement of the contacts; in the circumstances of the experiment, this method is not always possible, as it is of first importance that the plates be taken rapidly. While the positions of the paired contacts are drawn exactly alike in Figs. 2 and 3, it should be understood that these two figures are diagrammatic and that the exact replacement portrayed was certainly not achieved. The replacement was only approximate.

During the 20 minutes which the second period of flutter lasted the auricle beat regularly at a rate of between 345 and 352 per minute. Examples of the curves from lead *II* are shown in Figs. 23, 24, 25 and 26. The auricular complex is similar to that of the 1st after-effect, differing from it in two minor aspects; it is of somewhat lesser amplitude (this was due to a drop in the sensitivity of the recording instrument, which was not observed until later)*; the summit is more deeply notched.

S. A. N. lead (Record 17). The *Z* contact was placed below and the *C* contact above (see Fig. 3). The curve (Fig. 23) was similar to that previously obtained, the upright deflection indicating a movement of the excitation wave up the cava against the direction of the blood stream. The deflection preceded the summit of the auricular complex in lead *II* by 0.0374 of a second.

Mid-caval lead (Records 14 and 18). The *Z* contact was placed below and the *C* contact above, the pair being in the line of the tænia. The curve of record 18, which is representative, is shown in Fig. 25. The prominent downward deflections indicate the movement of the excitation wave down the tænia. The downstroke preceded the summit of the auricular complex by an average interval of 0.0237 and 0.0175 of a second, for the two plates,

* Note the larger ventricular complexes in the curves of the 1st after-effect (Figs. 21 and 22). The curves have been corrected in charting them in Figs. 5 and 6.

respectively. The curves were taken 1 minute and 7 minutes, respectively, after the beginning of flutter (see Table II).

Appendix lead (Record 16). The C contact was placed nearest the tip of the appendix, the Z contact being lower on the appendix and in line with it. The deflections were upright (Fig. 26) indicating movement of the excitation wave up the appendix towards its tip. The upstroke preceded the summit of the auricular complex by an average interval of 0.0096 of a second.

Inferior caval lead (Record 15). The Z contact was placed nearest the vein, the pair being in line with it. The deflections were downwardly directed (Fig. 24), indicating movement of the excitation wave down the vein against the direction of the blood stream. The downstroke followed the summit of the auricular complex by an average interval of 0.0053 of a second.

Fig. 5 is a chart summarising the readings: it has been constructed in similar fashion to Fig. 4.*

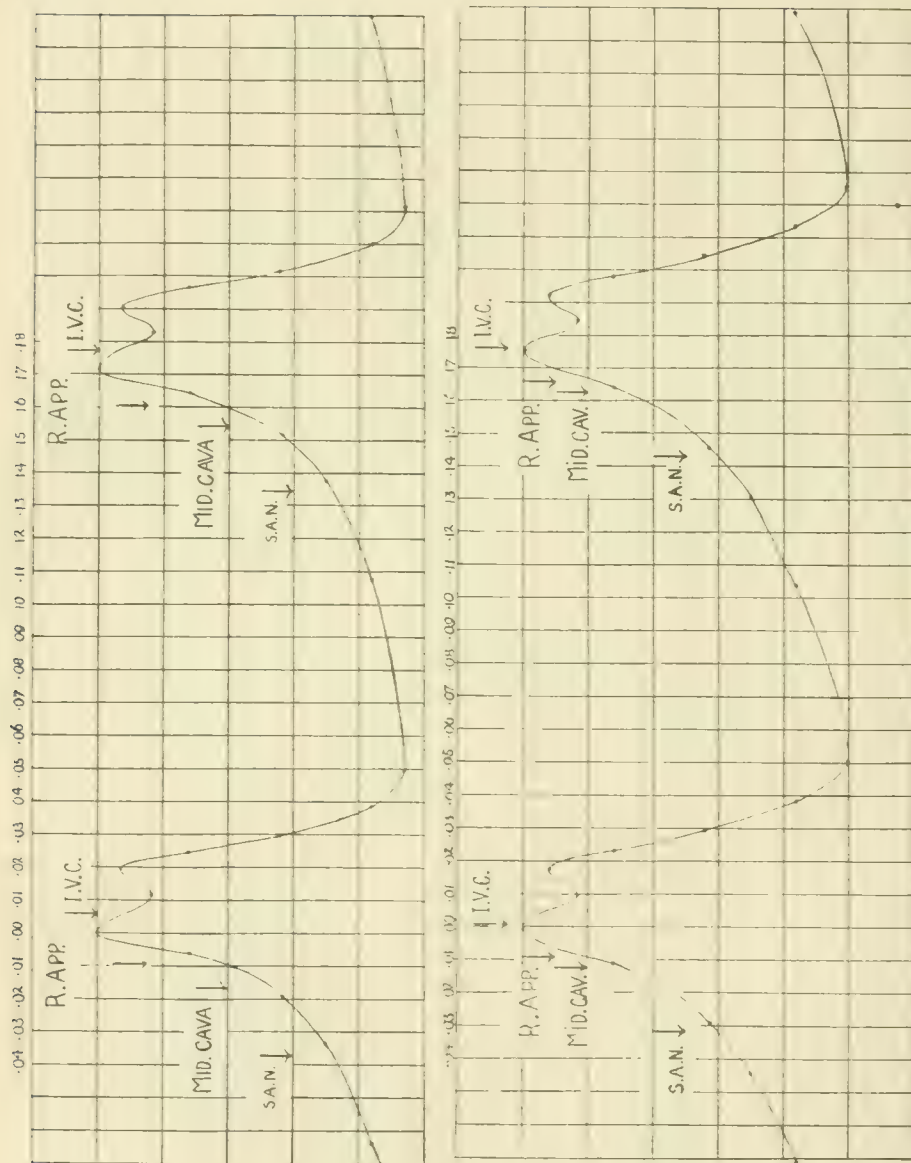
The flutter continuing, the leads from the four points of the auricular surface were repeated. The directions of the deflections were in all instances the same as those already described: the order of excitation also remained the same: there were, however, some minor changes in the readings relative to the summits of the auricular complexes (Fig. 6). The two series of readings of the 2nd stage are tabulated with those of the 1st stage, in Table II: the intervals between successive deflections are given in Table I.

Comments.

The experiment described illustrates certain of the main features of flutter, many of which are common to this and other experiments. The main conclusions to be drawn from it are these:—

1. In flutter, the separate parts of the auricular muscle contract in an orderly sequence, which is accurately repeated from cycle to cycle over considerable stretches of curve.
2. As might be expected from the last conclusion, the arrival of the excitation wave at any given point of the auricular muscle bears a definite relation to the auricular complex, as this is recorded in limb leads.
3. The direction which the excitation wave takes when flutter has become established is independent of the point from which this flutter was originally started. This third conclusion is one of some consequence, and we may state at once that we base it, not only upon the present experiment, but on many others where the after-effect was of shorter duration. In the present experiment the direction of spread was constantly from the base to the tip of the right appendix, although in one instance the stimulus which

* Variations from the average intervals as charted were as follows:—Record 15, + 0.0028 and - 0.0028; record 16, - 0.0008 and + 0.0009; record 17, + 0.0024 and - 0.0016; record 18, - 0.0011 and - 0.0008.



Figs. 5 and 6. *Dog J.P.* (Records 15-18 and 19-22, respectively.) These two charts are constructed in the same fashion as Fig. 4. They relate the times at which the excitation wave appeared at the surface points (see Fig. 3) to the auricular complex, during the first half (Fig. 5) and the second half (Fig. 6) of the second aftereffect shown by this animal.

TABLE I.
Leighs in seconds of successive auricular cycles in places taken from two separate after effects. (Dog J.P.)

Record Number	9 (S. J. A.) 1.5 sec.	10 (in App) 2.1 min.	11 (J. V. C.) 3 min.	12 (Mid-cava) 3.4 min.	14 (Mid-cava) 1 min.	15 (J. V. C.) 2 min.	16 (Rt. App.) 3.5 min.	17 (S. J. A.) 3.5 min.	18 (Mid-cava) 3 min.	19 (in App) 3 min.	20 (J. V. C.) 11 min.	21 (Mid-cava) 13 min.	22 (S. A. V.) 14 min.
	-1525 -1502 -1480 -1547 -1531 -1555 -1548 -1533 -1534 -1554 -1552 -1555 -1509 -1534 -1517 -1521	-1688 -1628 -1554 -1571 -1555 -1557 -1548 -1620 -1636 -1620 -1559 -1575 -1565 -1565 -1668 -1606	-1631 -1600 -1569 -1543 -1557 -1557 -1620 -1630 -1603 -1603 -1553 -1593 -1611 -1616 -1617 -1606	-1534 -1513 -1580 -1497 -1576 -1576 -1633 -1640 -1590 -1556 -1586 -1573 -1560 -1493 -1499	-1740 -1748 -1739 -1725 -1742 -1739 -1730 -1730 -1751 -1729 -1749 -1731 -1739 -1736 -1740	-1726 -1683 -1728 -1709 -1725 -1681 -1691 -1748 -1695 -1713 -1718 -1688 -1717 -1698 -1706 -1704	-1717 -1697 -1721 -1686 -1725 -1701 -1699 -1695 -1713 -1718 -1688 -1717 -1698 -1706 -1704	-1709 -1708 -1707 -1689 -1701 -1715 -1715 -1700 -1673 -1730 -1718 -1709 -1694 -1716 -1696	-1738 -1714 -1737 -1728 -1702 -1718 -1735 -1735 -1723 -1734 -1719 -1708 -1718 -1734 -1704	-1741 -1730 -1730 -1704 -1750 -1738 -1730 -1730 -1716 -1763 -1728 -1728 -1750 -1750 -1709	-1750 -1761 -1733 -1738 -1729 -1734 -1732 -1732 -1692 -1697 -1730 -1743 -1702 -1748 -1742 -1730 -1743	-1708 -1716 -1712 -1731 -1769 -1720 -1724 -1714 -1692 -1697 -1702 -1748 -1740 -1740 -1743	
Average	-1529	-1596	-1596	-1539	-1738	-1707	-1706	-1703	-1722	-1731	-1731	-1723	-1739
Max. Diff.	0075	0140	0088	0147	0026	0067	0039	0057	0036	0059	0078	0077	0048
Auricular Rate per minute	392	376	376	385	345	351	352	352	348	346	346	348	345

1st flutter lasted 6 min.

2nd flutter lasted 20 min.

TABLE II.

Readings of surface points in two separate after effects. (Dog J.P.)

	Point of auricle investigated	Record.	Summit of P to atrium.	Times of auricular deflection related to S. A. V. deflection.	Record taken after onset of flutter.
1st after effect, following stimulation of appendix.	S. A. V. region	9	0482	0000	15 seconds
	Mid-caval region	12	0249	0233	5 minutes
	Right appendix	10	0055	0427	2 minutes
	Inferior vena cava	11	0186	0668	4 minutes
	S. A. V. region (next beat)	9	..	1570	15 seconds
2nd after effect, following stimulation of inferior vena cava	S. A. V. region	17	0374	0000	5 minutes
	Mid-caval region	14	0237	0137	1 minute
	Mid-caval region	18	0175	0199	7 minutes
	Right appendix	16	0096	0278	4 minutes
	Inferior vena cava	15	0053	0427	2 minutes
	S. A. V. region (next beat)	17	..	1709	5 minutes
	S. A. V. region	22	0321	0000	14 minutes
	Mid-caval region	21	0125	0196	12 minutes
	Right appendix	19	0095	0226	8 minutes
	Inferior vena cava	20	0001	0322	11 minutes
	S. A. V. region (next beat)	22	..	1731	14 minutes

provoked the flutter had been applied to the tip of the appendix. The *general* direction in which the excitation wave moved through the body of the right auricle during the two periods of flutter was the same, though the one after-effect was provoked from the appendix and the other from the inferior cava. The original cause of the flutter consists of artificial stimuli entering a point in the auricular surface. The rapid beating is continued when the stimuli are withdrawn. We may conclude from this persistence that stimulation has set up a local or distant disturbance which continues, and that upon this disturbance the maintenance of flutter depends. But inasmuch as the direction of flow is not necessarily the same during the period of stimulation and during the period of the after-effect, we may conclude at the least that the disturbance set up is not confined to the region stimulated.

4. At the same time this experiment illustrates detailed variations in the after-effects. In the first place it is clear that, while the general direction of spread in the two after-effects described was the same, the detailed course was not the same. This is reflected in the minor differences in the auricular complexes as these appear in the curves taken from lead *II*: thus, during the first after-effect, a little notch precedes the main rise of the auricular complex (Fig. 4), while the notch at the summit is small, inconspicuous or absent: during the second after-effect (Figs. 5 and 6) the notch before the main upstroke is absent, and the summit is conspicuously and uniformly bifid. Corresponding differences in detail are to be seen in the readings from the surface: for while these are in general similar, fall in the same order, and are all related to the main upstroke and summit of the auricular complex (Figs. 4, 5 and 6), yet there is minor change, which is most notable in the region of the inferior cava.*

It is also apparent from the observations upon the second after-effect that the precise time relations may alter actually during the course of a single after-effect. The change is small and takes place slowly. Thus, during the course of this after-effect, three readings were obtained from the mid caval region: these were -0.0237 (at 1 minute from the onset), -0.0175 (at 7 minutes), and -0.0125 (at $12\frac{1}{2}$ minutes). Changes of this degree are insufficient materially to complicate our analysis. The variation in detail from after-effect to after-effect is so customary in different experiments, though it is not invariable, that it would obviously be unsafe to map out the excitation wave by means of data derived from more than one such after-effect. The variation during the course of a single after-effect is usually trifling, as this example illustrates: it is safe to compare readings from different regions, providing that the auricular rate remains constant and the auricular complex shows no distinct change.

* In the first period of flutter the wave proceeded more directly in the line of the inferior cava. It may be that in the first period this vessel was encircled by the wave, while in the second it was short-circuited (see observations on *Dogs JT* and *KQ*). This, as will become clear at a later stage, would be compatible with the slower rate of flutter during the first after-effect.

OBSERVATION 2. (*Dog JT.*)*First after-effect.*

In a dog of 5.7 kilogrammes weight the heart was opened in the usual fashion by splitting the sternum.

As a preliminary step, two pairs of contacts were placed upon the body of the right auricle and in line with the tip of the appendix. Each pair of contacts was connected to one string of the double fibre galvanometer. The appendix was stimulated by means of rhythmic induction shocks, so that excitation waves might be propagated in the line of, and over, the contacts and yield deflections as they passed over these contacts. The proximal contacts of the two pairs lay 9 millimetres apart, and the rate of conduction for the excitation wave was calculated for various speeds of rhythmic stimulation. These observations and the rates of conduction were tabulated for another purpose (see Part III). The rate which at present concerns us is that obtaining when the rhythmic shocks entered the appendix and gave response at a rate of 463 per minute. The corresponding rate in the auricular tissue was calculated at 520 millimetres per second.

The auricle was now stimulated at various points of its surface with rhythmic shocks at a rate approximating 700 per minute. At this rate of stimulation the auricle responded irregularly and continued to beat for variable periods after the withdrawal of the stimulating electrodes. Usually the after-effect lasted a few seconds only. On one occasion, however, on stimulating the inferior vena caval region, an after-effect, lasting 30 minutes, was obtained. It is with this long-lasting after-effect that the present description is concerned.

The after-effect, as seen in curves from lead *II*, consisted of auricular and ventricular complexes, the former placed regularly, the latter somewhat irregularly. The auricular complexes were of uniform shape, and consisted each of a relatively steep upstroke, and an almost equally steep downstroke, terminating in a gradual ascent to the next upstroke (Figs. 27-31). The complexes were therefore contiguous, in the sense that no true isoelectric period separated them.* In any direct lead from the auricle, the deflections were uniform in direction, outline and amplitude as they were in the last experiment, thus demonstrating the passage of the excitation wave in a constant direction across any given pair of contacts. The excitation waves appeared in a regular rhythm, the regularity being of a high order. The regularity may be illustrated by the fine measurements of succeeding cycles in these curves (see Table III). The degree of irregularity shown by these figures is little greater than the probable error of measurement, with the exception of record 19, in which the irregularity is slightly greater.

* In the curves these complexes are distorted by falling with portions of the ventricular complexes: the description is based upon an analysis of the curves which takes the simultaneous events of the ventricle into account (see Fig. 9).

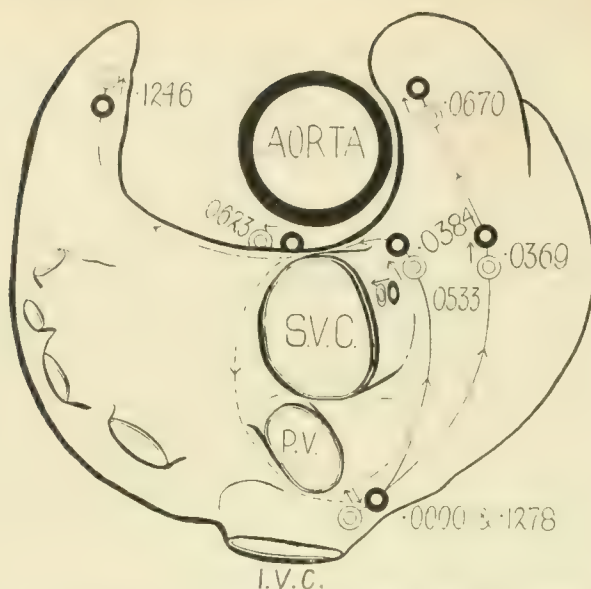


Fig. 7. *Dog JT.* (Records 13-19.) An accurate outline of the cephalic surface of a dog's auricle, somewhat enlarged. The arrangement of the contacts used in the direct leads is shown, the Z contact being drawn black and the C contact white. The short arrows represent the path of the excitation wave past the individual contacts, as indicated by the direction of the intrinsic deflection. The long unbroken arrows represent the general course of the excitation wave, as ascertained both by the direction of deflections and the readings taken from the surface. These readings, given in decimal points of a second, have been reduced to a new common zero (see Table IV). The broken arrow, lying to the left of *S. V. C.* and *P. V.* in the figure, indicates the hypothetical course of the wave to complete its central circuit. *S. V. C.*=superior vena cava; *P. V.*=right pulmonary veins.

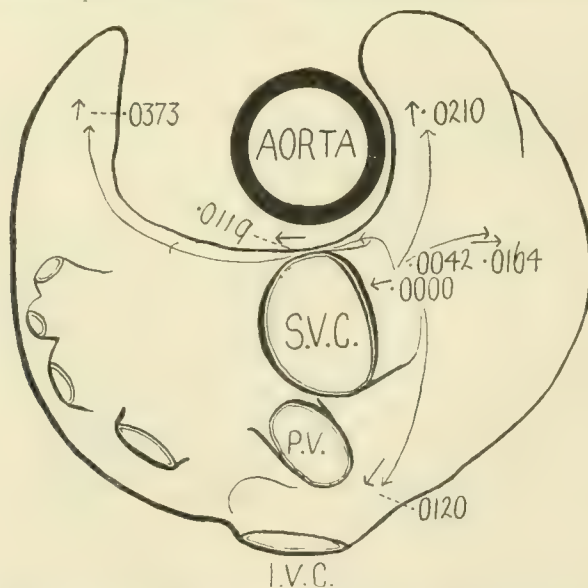


Fig. 8. *Dog JT.* (Records 25-31.) A second outline of the same heart, examined in the same fashion during the progress of the normal heart rhythm. The readings were obtained relative to the summit of *P* in the electrocardiogram (see Fig. 10), but have been reduced to a new common zero, namely, the reading from the base of the superior vena cava. The short arrows indicate the path of the excitation wave, as judged by the direction of the deflections. The long arrows show the general path as ascertained from these data and the surface readings.

TABLE III.

Lengths in seconds of successive auricular cycles in seven plates. (Dog J.T.)

Record No.	Base of rt. appendix 13 ($\frac{1}{2}$ min.)*	Tip of rt. appendix 14 (2 mins.)	Inferior vena cava 15 (4 $\frac{1}{2}$ mins.)	S. A. N. region 16 (7 mins.)	S. V. C. 17 (10 mins.)	Band 18 (14 mins.)	Left appendix 19 (23 mins.)
	·1145	·1228	·1228	·1270	·1289	·1310	·1268
	·1144	·1237	·1215	·1280	·1306	·1305	·1261
	·1132	·1214	·1223	·1262	·1295	·1303	·1281
	·1157	·1228	·1205	·1260	·1283	·1288	·1302
	·1131	·1234	·1208	·1256	·1301	·1305	·1261
	·1153	·1227	·1217	·1269	·1292	·1311	·1302
	·1141	·1225	·1214	·1276	·1297	·1294	·1272
	·1137	·1226	·1213	·1260	·1302	·1305	·1310
	·1141	·1221	·1212	·1272	·1283	·1293	·1272
	·1156	·1223	·1207	·1255	·1299	·1311	·1328
	·1130	·1220	·1226	·1282	·1288	·1310	·1255
	·1155	·1235	·1215	·1271	·1301	·1290	·1326
	·1134	·2229	·1216	·1267	·1289	·1300	·1303
	·1142	·1231	·1221	·1273	·1290	·1294	·1282
Average	·1143	·1227	·1216	·1269	·1294	·1301	·1287
Max. Diff.	·0027	·0023	·0023	·0027	·0023	·0023	·0073
Auricular rate per minute	52.5	499	493	473	464	461	466

* Time after onset of flutter.

During the progress of the flutter curves were taken by means of direct leads from various points on the surface of the auricle. They were taken separately and each simultaneously with a curve from lead *II*, the latter being used as a standard of measurement.

Base and tip of right auricular appendix (Records 13 and 14). A pair of contacts placed at first on the body of the auricle at the base of the right auricular appendix and in line with the appendix, and later on the right appendix itself (the *Z* contact being on both occasions towards the tip of the appendix) (Fig. 7), showed prominent and uniform intrinsic deflections (having a downward direction in the curves, see Fig. 27), indicating the passage of excitation waves up the body of the auricle in the line of the contacts, and from the base to the point of the appendix (see arrows in Fig. 7). The intrinsic deflections recorded from these two points appeared regularly

at time intervals of 0.0516 and 0.0215 of a second,* respectively, before the summits of the corresponding auricular complexes in lead *II* (see Table IV).

S.A.N. region (Record 16). In a curve from a pair of contacts placed on the upper part of the tænia terminalis (the *Z* contact being placed above and the *C* contact below), uniform, intrinsic deflections, directed downwards in the curve, were displayed: they indicated a movement of the excitation waves up the tænia in a direction more or less in the line of the contacts. These deflections appeared regularly at intervals of 0.0501 of a second before the summits of the corresponding auricular complexes in lead *II*.

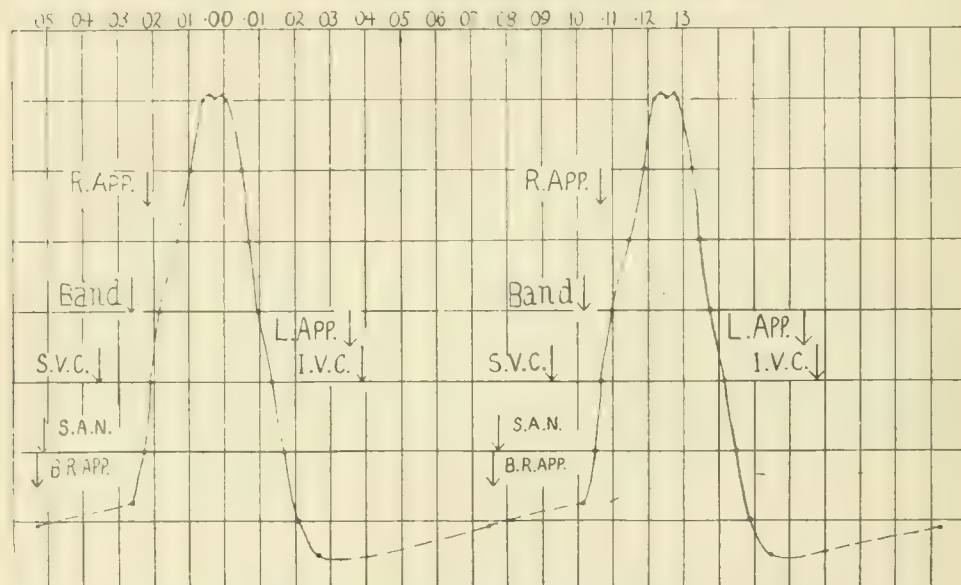


Fig. 9. *Dog J.T.* (Records 12-19.) A chart constructed in the same fashion as Fig. 4. The auricular complex measured was taken from Record 18. The chart displays the relation of surface readings to the auricular complex during the period of flutter. The voltages of that portion of the line which is broken are somewhat uncertain, because the auricular complexes were falling with the ventricular complexes and suffering distortion. Ordinates = one-tenth millivolt. Abscissæ = 0.01 of a second.

Intra-auricular band (Record 18). A curve was obtained by special curved electrodes consisting of a pair of contacts inserted behind the aorta, the contacts lying on the intra-auricular band and in the line of it; the *Z* contact lay to the right and the *C* contact to the left. The intrinsic deflections were uniform, prominent and upright (Fig. 29), indicating the passage of the excitation waves from right to left across the band in the line of the contacts. These deflections appeared regularly at an interval of

*The times at which successive deflections in a direct lead appeared relative to the summit of the corresponding auricular complexes showed no greater variation from the value charted (in Fig. 7) than 0.0032 of a second. The maximal variations in intervals on the seven records were:—Record 13, +.0012 and —.0012; Record 14, ±.0000; Record 15, +.0008 and —.0017; Record 16, +.0015 and —.0017; Record 17, +.0015 and —.0022; Record 18, +.0005 and —.0004; Record 19, —.0032 and —.0027. Such variation as occurs is in the whole series no greater than the error of measurement, with the exception of the measurements of Record 19.

0.0262 of a second before the summits of the corresponding auricular complexes in lead *II*.

Superior vena cava (Record 17). A pair of contacts placed on the superior cava and in line with it (the Z contact being below and the C contact above) yielded prominent, uniform and upright deflections, indicating the passage of the excitation waves up this vessel in the line of the contacts (Fig. 28). These deflections appeared regularly 0.0352 of a second before the summits of the corresponding auricular complexes in lead *II*.

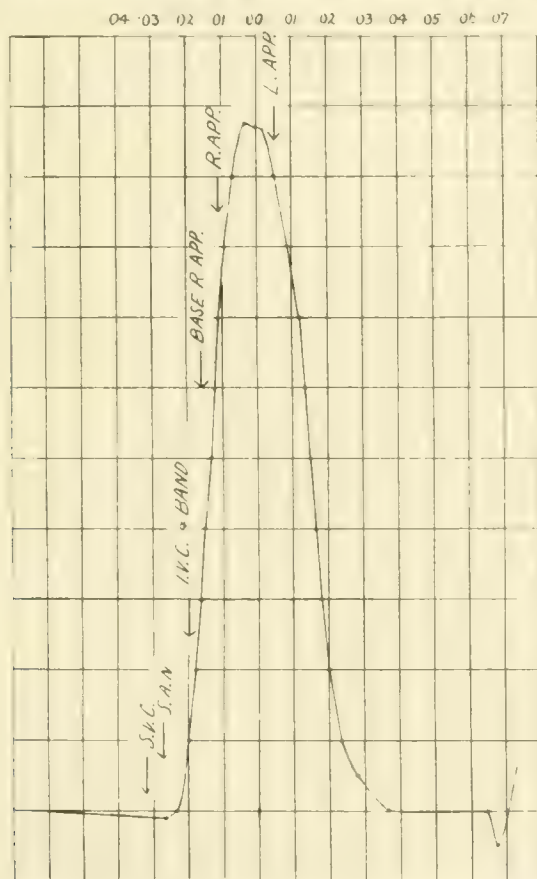


Fig. 10. Dog J.T. (Records 25-31.) A similar chart to the last. It displays the relation of surface readings to the auricular complex while the heart beats normally. Ordinates = one-tenth millivolt. Abscissa = 0.01 of a second.

Inferior cava (Record 15). A pair of contacts was placed on the base of the inferior cava and in line with this vessel (the Z contact being placed nearer to the mouth of the vessel). The deflections were uniform but small (Fig. 31), tending to show that the excitation waves were passing at an obtuse angle to the line of the contacts. The chief deflections (intrinsic) began regularly 0.0885 of a second before the summits of the corresponding auricular complexes in lead *II*.

Left appendix (Record 19). A pair of contacts placed on the inner surface of the left appendix and in line with it (the *Z* contact being placed away from the tip of the appendix), gave large and uniform intrinsic deflections, whose upward direction in the curves (Fig. 30) indicated the passage of the excitation waves towards the tip of the appendix in the line of the contacts. It was necessary to move the heart to obtain this, the curve from the last lead, and as a consequence the form of the auricular complex in lead *II* altered a little. This alteration of shape rendered the position of the original summit of the auricular complex (the standard of measurement) somewhat doubtful.* The intrinsic deflections appeared constantly 0.0361 of a second *after* the probably correct summits in lead *II*.

The data, so far described, are summed up in Figs. 7 and 9. The diagram (Fig. 7) is an accurate drawing of the cephalic surface of a dog's auricle, and shows as accurately as has been possible the positions of the paired contacts employed, and their distances from each other.† The *Z* contact is drawn as a black and the *C* contact as a white circle. The short arrows drawn in firm lines show the direction travelled by the excitation wave, as indicated by the direction of the observed deflections. The long unbroken arrows indicate the direction taken by the excitation wave, as ascertained from the direction of the deflections on the one hand and the readings from the surface on the other.

Fig. 9 is a chart constructed in a similar fashion to that of Fig. 4, etc. Now it will be apparent from the chart that the deflection in the inferior caval lead may be taken as following the auricular summit in lead *II* by 0.0393 of a second; it may also be taken as preceding the next auricular in lead *II* by 0.0885 of a second (see Fig. 31). Stated differently, if we take the deflection of the inferior caval lead as an arbitrary zero point, it appears again at 0.1278 of a second, this being the time during which our charted auricular cycle is completed.

The times at which the excitation wave was ascertained to arrive at the several direct contact points, relative to the summit of the auricular complex in lead *II*, are tabulated in the third column of Table IV. The fourth column of the same table assumes the time of arrival at the inferior caval contact to be the zero (as opposed to the summit of the auricular complex), and relates the remaining deflections to this new time instant. The object of this translation to a new zero point is purely to render description clearer. The readings, reduced to this new common zero, have been entered in the diagram of Fig. 7.

* Even if the wrong point of measurement has been chosen the error introduced is not great.

† The heart of the dog actually used in the experiment was unfortunately not available when the diagram came to be constructed. The heart of a dog of approximately the same size has therefore been employed. At the time of the experiment the positions of the contacts was noted accurately in relation to surrounding landmarks and were sketched in a diagram which was not accurately to scale. The importance of keeping the heart for measurement was not appreciated when the experiment was performed. The errors thus introduced are not large.

The known course of the excitation wave

The direction of the intrinsic deflections has already shown us the direction in which the excitation wave was travelling during this auricular flutter at the actual contact points. The direction was up the body of the right auricle towards the tip of the appendix, up the tænia, up the superior cava, from right to left across the intra-auricular band and towards the tip of the left appendix. The times at which the intrinsic deflections appeared, relative to each other, fully confirm these conclusions and extend them. The movement was from inferior cava upwards over the body of the auricle and along the right auricular appendix. Arriving at the upper part of the tænia, the wave turned up the superior vena cava and also proceeded, via the intra-auricular band, to the tip of the left auricular appendix.

TABLE IV.

Actual and calculated readings for the arrival of the excitation wave at seven points of the auricular surface, during auricular flutter. (Dog J.T.)

Point of auricle investigated.	Record No.	Summit of P to a definite deflection (averages)	Times of intrinsic deflections related to I. V. C. deflection.	Calculated times at which intrinsic deflections should occur (in seconds)	Approximate distance of point from I. V. C. in millimetres.	Actual auricular rate.
Inferior vena cava	15	— 0885	0000	0000	0	493
Base of rt. appendix	13	— 0516	0369	042	22	525
S. A. N. region	16	— 0501	0384	042	22	473
Superior vena cava	17	— 0352	0533	050	26	464
Inter-auricular band	18	— 0262	0623	065	34	461
Tip of rt. appendix	14 (20)	— 0215 — 0133	0670 0752	073	38	490
Left appendix	19	+ 0361	1246	117	61	466
Inferior vena cava (next deflection)	15	+ 0393	1278	129	67 (circuit)	493

* On basis of observed conduction rate of 520 millimetres per second (auricle responding to stimulation at rate of 463 per minute).

The auricle was beating at a rate of between 461 and 525 (see Table IV) per minute; the observed rate of conduction while this auricle was beating in response to rhythmic stimulation at a rate of 463 per minute was 520 millimetres per second, as previously stated. Now the distances which an excitation wave, starting at the inferior cava and travelling by the paths indicated, would have to travel are known approximately from measurement (Fig. 7). These measurements are included in the last columns of Table IV. Using these figures, and the conduction rate of 520 millimetres per second, we may calculate the times at which the excitation wave would reach the

various contact points examined, assuming the wave to travel along straight paths in the directions indicated. These calculated times are given in the fifth column of Table IV. Considering the possibilities of minor error in this computation, the correspondence between the figures in the fourth and fifth columns is so close as to be beyond the possibility of coincidence.

The hypothetical path.

The observations so far detailed clearly establish the course of the excitation wave over the surface of the right auricle and over the surface of the left auricle exposed in the experiment. It remains to inquire its course through the remainder of the auricle. The whole auricular cycle lasted 0.1278 of a second,* the journey from inferior cava to a point 15 millimetres short of the tip of the left appendix† occupying 0.1246 of a second. The path was from the inferior cava, up the tænia, through the band and from base to apex of the left appendix. It is clear therefore, from the readings taken on this path, that some new portion of the auricular tissue became active during each phase of the auricular cycle and that there was no true diastole. It might be assumed that impulses arose in the region of the inferior cava and spread over the whole auricle at a slow rate to end finally at the extreme tip of the left appendix. But another explanation is possible, and more fully explains our observations. It is that the excitation wave travelled back to the inferior cava by a separate path and that a second and similar movement of the same excitation wave was started.

If we assume the excitation wave to have re-entered the circuit at the inferior cava by a separate path from the left auricle, we must assume the shortest path. This path is not via the left appendix, but via the auricular tissue lying in our figure directly to the left of the superior vena cava and right pulmonary veins, the path indicated by the dotted line (Fig. 7). It is a path which, from the contact on the intra-auricular band to that on the inferior cava, had a length of approximately 34 millimetres; the time calculated for the wave to travel along this path is 0.065 of a second: the interval between the appearance of the excitation wave at these two contacts was actually found to be 0.0623 of a second. To describe the same thing somewhat differently, the length of the whole circuit from the inferior cava round the superior cava and back to the inferior cava was approximately 67 millimetres; the calculated time for the wave to travel around this circuit is 0.129 of a second; the length of the whole auricular cycle was observed at 0.1278 of a second.

* This was the duration of the whole auricular cycle in lead *II*, which is charted in Fig. 7. The average length of auricular cycle for different plates is given in Table III; it lay usually between 0.1216 and 0.1294 of a second (for the first point examined the duration was shorter, *i.e.*, 0.1143 of a second). The value 0.1278 sufficiently expresses a middle value, and its use introduces no material error.

† We say 15 millimetres short of the tip because the proximal contact lay at about this distance from the actual tip.

This experiment presents strong presumptive evidence of a circus movement in the auricles, a movement taking place around a natural ring of muscle, formed mainly by the great orifice of the superior vena cava. The direction of movement was anti-clockwise through the muscle as this ring is viewed from above.

In this same animal, the times at which the excitation wave arrived at the chief contact points investigated were ascertained while the heart beat naturally (Fig. 8).

They are shown in Fig. 8, the lowest reading, namely, that obtained over the region of the sino-auricular node, being taken as the zero. The order of activation is the usual order for the dog's auricle (see Lewis, Meakins and White¹). The readings are of interest in the present connection, first, because they contrast in order with those obtained during the period of flutter. The spread, while the heart beat normally, was radial to the pacemaker: the wave flowed to the left through the band to the left appendix, to the right down the tænia terminalis, and forwards through the body of the auricle to the right appendix. They are also of importance because, when the rates of conduction are calculated, these contrast with those prevailing during the phase of flutter. Thus, the time taken for the wave to travel from the S.A.N. region to the left appendix was 0.0373 of a second during the natural heart beat: it was 0.0862 of a second during the period of flutter. The interval between the appearance of the excitation wave at the S.A.N. region and the inferior vena cava was 0.0120 during the natural heart beat: it was 0.0384 during the period of flutter.* Thus, the transmission rate during flutter would appear from these isolated calculations to be about half (or one-third) the natural rate.

It is of interest also to compare the relation of the surface readings to the auricular complexes. In Fig. 10, as will be seen, the intrinsic deflections all fall on the upstroke of the auricular complex, in relation to the short stretch of curve which precedes the upstroke and to the summit itself. Now this illustrates a general rule, when the heart beats normally. The latest points activated show intrinsic deflections which fall at or about the summit, or perhaps a little later. At this point the excitation has reached† all parts of the auricle: the activation is crowded into the short space of a few hundredths of a second. In flutter the activation of the auricle takes longer: and, as the auricular cycle is much shorter, it is spread over the whole cycle.

* The last comparison is perhaps not so valuable, in that the excitation wave was travelling in contrary directions on the two occasions.

† We say "reached" advisedly.

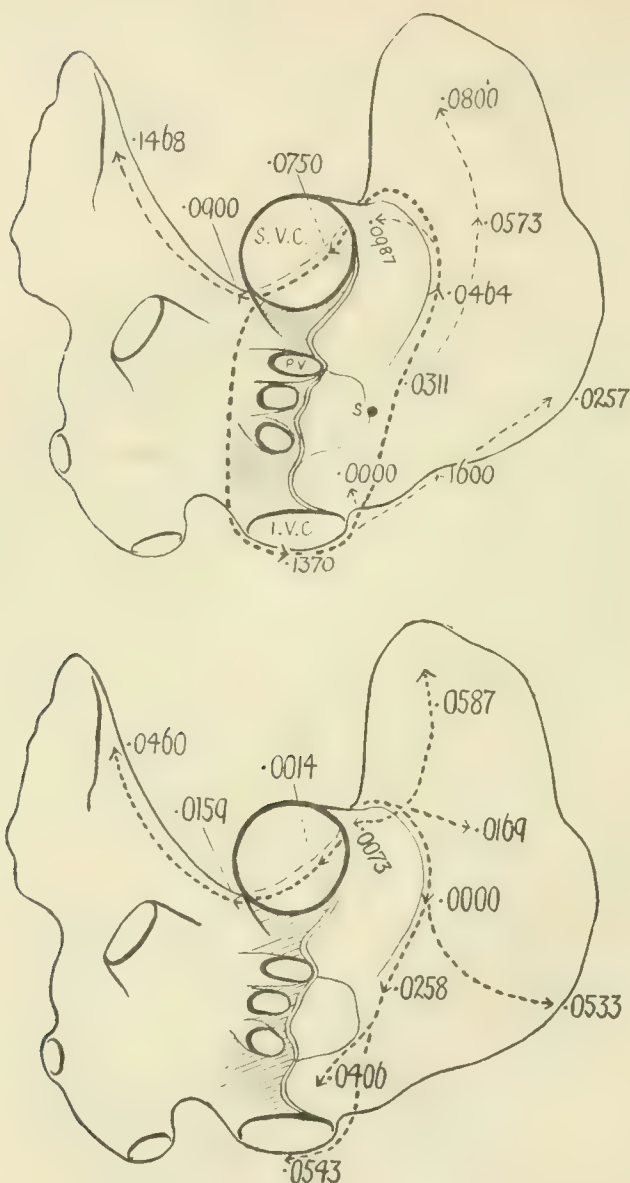


Fig. 11. *Dog KQ*. (Records 29-41.) An accurate and natural size outline of the auricle of *Dog KQ*, showing the readings obtained during the period of flutter. These have been reduced to a new common zero (see Table V and Fig. 13). The broken arrows represent the path pursued by the excitation wave, as ascertained from the direction of the deflections in direct leads and from the surface readings; the former and latter evidences were entirely confirmatory of each other. An area is shaded to display more prominently the pericardial reflection. S.V.C.= superior and I.V.C.= inferior vena cava. P.V.= right pulmonary veins. S marks the point stimulated.

Fig. 12. *Dog KQ*. (Records 42-43.) The same auricle, examined while the heart beat naturally.

Second after-effect.

The period of flutter which has been discussed and which was induced by stimulating the *inferior vena cava*, lasted 30 minutes and ended. Stimulation of the appendix failed to re-induce flutter, but stimulation of the *upper part of the sulcus terminalis* yielded an after-effect of 3 minutes' duration. This after-effect, as studied in lead *II*, resembled very closely the flutter seen in the last stages of the first and long period of flutter. Two direct leads were obtained from the heart.

Right appendix lead (Record 21). The curve from this lead (with *Z* contact below and *C* contact above) showed that the excitation wave was moving towards the tip of the appendix; the intrinsic deflections were prominent deflections, uniform in sequence, form and directed upwards. The deflections preceded the summit of the auricular complex in lead *II* by 0.0259 of a second. During the long period of flutter the figures for the right appendix were 0.0215 and 0.0133, respectively (Records 14 and 20, Table IV.).

S.A.N. lead (Record 22). The curve was taken with the *Z* contact above and the *C* contact below. The intrinsic deflections were uniform in sequence and form, being directed downward and thus showing a movement of the excitation wave up the tænia. The deflections preceded the auricular summits in lead *II* by 0.0632 of a second. During the long period of flutter the reading for this region was 0.0501 of a second.

Comment. The foregoing after-effects, the first lasting 30 minutes and excited from the inferior cava, the second lasting three minutes and excited from the upper end of the sulcus, were very similar. The general order in which various different regions of the auricle became active was the same during the two periods. This is shown, because (1) the auricular curves from lead *II* in the two periods were similar; (2) the direction taken by the excitation wave in the appendix (being from base to tip) and in the tænia (being from below upwards) was the same in both instances. This is indicated by the direction of the intrinsic deflections, seen in direct leads from these regions; (3) the time relations of the intrinsic deflection obtained from the *S.A.N.* region and from the appendix to each other were similar, and the time relations of these to the auricular complex in lead *II* showed no notable variation in the two periods. The experiment again exemplifies what has been shown, namely, that the order in which the muscle becomes activated during the progress of flutter is independent of the region from which that flutter is originally excited.

OBSERVATION 3 (Dog KQ.)

In a dog of 15 kilogrammes weight a pure flutter lasting 35 minutes was produced by means of rhythmic stimulation of the mid-caval region at the rate of 573 per minute. This flutter was examined in the usual way, and it proved to be very similar to that observed in the last experiment. It is published as a confirmatory experiment. It will be described more briefly, as it seems unnecessary to enter again into complete detail. An example of the curves is shown in Fig. 33. The actual readings of the surface points relative to the auricular complex are shown in Table V and in Fig. 13, which has been constructed in the same fashion as Fig. 4, etc. The reading for the surface points have been reduced to a new zero (see the fifth column of the table), namely, the time instant at which the *I. V. C.* became active, and these new readings have been charted upon a natural sized scale drawing of the corresponding auricle; the latter is shown in Fig. 11. The course taken by the excitation wave during this flutter is indicated by the broken lines and arrows of this figure, and these have been filled in after considering (*a*) the direction of the intrinsic deflections, indicating the contact of the pair first experiencing active muscle beneath it, and (*b*) the order of the actual readings. As the detailed description of the leads and curves is omitted, it is to be stated that these data were in full agreement. The heavy broken line indicates the circuit followed by the excitation wave; that part of it which lies to the right of the great vessels in the diagram and above the superior cava having been directly ascertained, that which lies to the left of the pulmonary veins and inferior cava in the diagram being to an extent hypothetical.* Now the whole length of the surface muscular track from inferior cava along the *tænia*, around the superior cava and back around the right pulmonary veins and inferior cava measured 137 centimetres. The whole length of the auricular cycle measured approximately 0.1600 of a second. The rate of conduction, assuming a circuit movement, is therefore calculated at 856 millimetres per second. Using this calculated conduction rate, and knowing the approximate distance, we may estimate the times at which the excitation wave would be anticipated at the various contact points on the circuit, and compare these with the actual readings. The readings in the two columns of Table V show very close approximation throughout, the greatest divergence being about 0.0150 of a second. The time at which the excitation wave is expected to arrive at the lower surface of the inferior cava on its return from the superior cava agrees almost precisely with the actual reading (the figures are 0.1366 and 0.1370 of a second), a close correspondence which is emphasised by the agreement of the actual and calculated readings for the region of the intra-auricular band (the figures

* The extent to which it is hypothetical depends upon its length: as the gap between contacts from which readings are taken is reduced, so the conclusion that the wave passes by the shortest path from one to the other (always providing that the readings are compatible with the conclusion) becomes less hypothetical and more certain. In the present instance the unexamined gap to the left is a smaller fraction of the circuit than in Fig. 7.

TABLE V.

Actual and calculated readings for the curved of the variation wave at points of the auricular surface during auricular flutter.
(Day KQ.)

Point investigated.	Record No.	Mins. after onset of flutter.	Summit of <i>P</i> to intrinsic in sec.	Intrinsic deflections related to <i>P</i> , <i>F</i> , <i>C</i> , intrinsic in sec.	Calculated times at which intrinsic deflections should occur.*	Distance of point from <i>P</i> , <i>F</i> , <i>C</i> , in millimetres.	Auricular rate.
Inferior vena cava (upper)	32	9.0	-0.45	-0.000	-0.000	0	380
A V groove (right)	33	10.2	-0.788	-0.257			369
Mid caval region	34	7.9	-0.734	-0.311	-0.200	17	374
S. I. V. region	30	6.2	-0.581	-0.464	-0.410	35	374
Base of right appendix	34	11.3	-0.472	-0.573			370
S. I. C. (left surface)	37 38	18.3 19.0	-0.295 -0.271	-0.750 -0.774	-0.640	55	368 369
Tip of right appendix	29	5.1	-0.245	-0.800			371
Band	39	19.7	-0.145	-0.900	-0.880	75	371
S. I. C. (up the vein)	35	12.7	-0.058	-0.987			372
Inferior vena cava (lower)	36	14.9	-0.225	-1.370	-1.366	117	368
Left appendix	40 41	23.5 33.3	-0.123 -0.111	-1.468 -1.359			378 371
Inferior vena cava (upper) (next deflection)	32	9.0	-0.555	-1.600	-1.600	137	380

* On a basis of a measured extent of 137 millimetres being completed during the time of a full auricular cycle (0.1600 of a second).

are 0.088 and 0.0900 of a second). This result obtained in a second experiment offers the strongest support to the view that auricular flutter consists essentially of a circus movement. In other respects the experiment confirms those already detailed.

The point originally stimulated was the mid-caval region. During the period of flutter the excitation wave travelled from the inferior cava towards the mid-caval neighbourhood.

On the superior cava and in the right and left appendices the movement of the excitation wave was centrifugal from the body of the auricle.

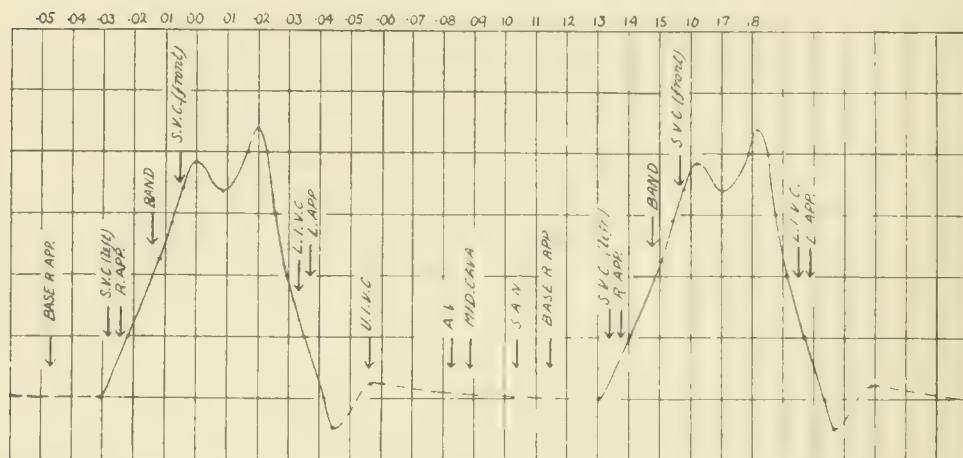


Fig. 13. *Dog KQ*. (Records 29-41.) A chart constructed in the same fashion as that shown in Fig. 4, to display the time relations of the intrinsic deflections to the auricular complex of lead *II* during the period of flutter. The voltages of that portion of the curve which is broken are somewhat uncertain, because the auricular complexes fell with ventricular complexes and suffered distortion as a consequence. Ordinates = one-tenth millivolt. Abscissæ = 0.01 of a second.

In the chart (Fig. 13) relating the intrinsic deflections to the auricular complex of lead *II* these intrinsic deflections are not confined to a short section of the cycle, but are diffused throughout it, as they have been in other instances of flutter. This relation shows, as has been stated, that some new part of the auricular muscle is being activated throughout the whole of the auricular cycle.

A comparison of this chart with that of Fig. 9 is particularly valuable in that it controls the accuracy of the two series of observations. In both instances we are dealing with flutter supposed to depend upon an anti-clockwise circus movement around the superior cava and pulmonary veins; it is true that in the first instance the inferior cava appears to be short-circuited, while in the last it is surrounded; but that is a matter of detail. The general, and in greater part the detailed, relation of the intrinsic deflections to the main upstroke and downstroke of the auricular complex of lead *II* is the same in

the two animals. Thus, the base of the right appendix becomes active a few hundredths of a second before the beginning of the main upstroke; the S. A. N. region is activated at about the same time instant in both, the intra-auricular band and the tip of the right appendix are both activated during the early part of the upstroke,* while the left appendix and inferior cava are activated near the end of the main downstroke. Such correspondence proves the substantial accuracy of the surface readings as a whole.

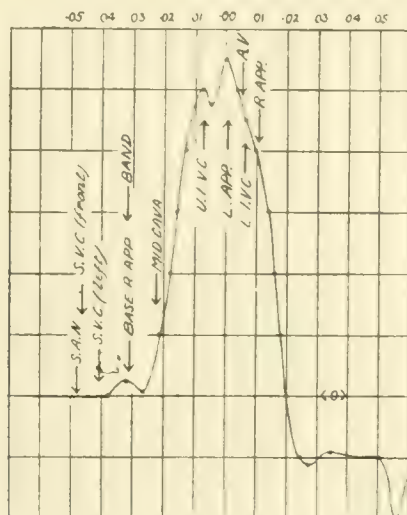


Fig. 14. *Dog KQ.* (Records 42-53.) A chart similarly constructed to display the relations of intrinsic deflections to the auricular complex, while the normal heart rhythm prevailed. Ordinates=one-tenth millivolt. Abscissæ=0.01 of a second.

The readings from the same points, while the heart of this animal beat naturally, are shown in Fig. 12, in which the broken lines and arrows indicate the direction taken by the excitation wave in its spread, estimated from the readings and the directions of the deflections, which were perfectly consistent. The corresponding chart, relating the intrinsic deflections to the *P* wave in lead *II*, is shown in Fig. 14. The crowding of these deflections upon the opening phases and summit of the curve as here shown is, as we have seen, customary when the auricle beats normally. It is unusual for points such as the tip of the left appendix and inferior cava (both of which are activated relatively late) to extend much beyond the actual summit.†

The observations during the progress of the natural heart beat are interesting also because they allow the natural conduction rates and those calculated for the period of flutter to be compared. This comparison is

* There are naturally differences in the finer details.

† Usually, indeed, they come a little earlier in relation to the summit than was the case in this animal.

soundest upon the intra-auricular band and left appendix; for here the wave pursued the same path on both occasions. Thus, during the period of flutter, the transmission time from the left of the superior cava (0.0750) to the left appendix (0.1468) was 0.0718 of a second; while during the natural beat it was 0.0446 of a second. This would indicate approximately a 60 per cent. increase in conduction rate when the heart returned to a natural rhythm. On the other hand, a comparison of the figures over the tænia does not show so material a difference. During the period of flutter the time taken for the wave to travel from lower inferior cava to *S.A.V.* region was 0.0694 of a second; while the heart beat naturally the interval was 0.0543 of a second. During the period of flutter conduction was depressed (the actual rate being about 856 millimetres per second) as compared to the normal for this animal, but the depression was not so great, neither did it actually reach so low a figure, as was the case of the experiment previously described (the conduction rate in that experiment was about 520 millimetres per second). The reason for this divergence is to be found in the rates of the flutter in the two animals. In the present experiment it was 370 per minute, in the former experiment it was 460-520 per minute. The rate of beating in auricular flutter is controlled, according to our views, by two factors, namely the state of conduction on the one hand and the length of the circuit on the other. A long circuit or a slow conduction rate will produce slow flutter rates. If that is so, then, *cæteris paribus*, we should anticipate that in larger animals the rate of flutter would be slower than in smaller animals; to institute a thorough comparison would require a greater number of complete experiments than we have at present at our disposal; in the absence of such we can but compare individual experiments in which the circuit appears to have been established in similar regions of the heart. It may be that in this relation of flutter rate to length of circuit is to be found the reason why in the dog flutter rates are so much higher than in man. In the dog the rates range from 345 to 580 beats per minute; in the human subject the known range is from 200 to 350 beats per minute.

OBSERVATION 4. (*Dog KW.* WEIGHT 11.1 KILOGRAMMES.)

In the experiments which have been described, convincing as they seem to us, we realise that there is a single point of weakness. No doubt remains as to the course taken by the excitation wave during the flutter in the experiments which have been discussed, in so far as the exposed surface of the right auricle, the left surface of the superior cava, the band and left appendix are concerned. The weakness obviously lies in the length of the gap between the intra-auricular band and the inferior cava on the return journey. It has seemed a most desirable thing to reduce this hiatus, to obtain a reading from the left auricle in the region where the right pulmonary veins spring from it. If a reading intermediate between the *band* and *inferior caval* readings were obtained from this region, any existing doubt

would clearly be dissolved. But this region of the auricle lies outside the reflections of the pericardium, and consequently a different method of exposing the heart to that adopted for the surface of the right auricle is needed. The whole surface of the right auricle, the band and the tip of the left appendix can be explored when the heart is slung in the opened pericardium, without altering the position of the heart during the experiment: but this exposure is not convenient if the body of the left auricle is to be tested. If readings have been obtained from the right auricle and band during a period of flutter, it might be thought possible, once these were completed, that the second exposure might be proceeded with and that readings from the left auricle might be added. For two reasons such attempts cannot often prove successful. First, because handling the heart in any way, once flutter has established itself, usually brings the flutter to an end, and, secondly, because, even if the flutter continues, displacement of the heart affects the form of the curve obtained from lead *II*, and thus confuses the standard from which measurements are subsequently made.* It might be possible to obtain the desired readings by leading from the internal surface of the auricle through a right pulmonary vein. This method has actually been tried on several occasions, though so far without success.

Meanwhile there is a less direct method of solving the difficulty. To take such an experiment as that last described, there can be but two views as to the meaning of the observations. The excitation wave is traced back to the region of the inferior cava as far as it can be traced: it either starts in this vicinity or reaches this part of the auricle from the left auricle. In the last case it is a circus movement, for if it can be definitely inferred that the readings are somewhat earlier on the left auricle than on the cava, the gap becomes of insufficient length to leave room for any reasonable doubt as to the nature of the condition. The first case, however, is still within the bounds of possibility: but that it is the true explanation is extremely improbable, since it would not account for that want of relation between the direction of movement during flutter and the point stimulated to induce it.

The following experiment has been devised to test the matter from a different point of view. It inquires if, when the inferior vena cava is stimulated by means of rhythmic induction shocks at a rate compatible with that prevailing in flutter, the readings from the surface of the auricle, obtained while the auricle is responding to this stimulation, correspond with those seen in flutter. The answer is in the negative. When the inferior cava is thus stimulated (in the illustration chosen the rate of stimulation and of response was 446 per minute) the excitation wave spreads from it

* The experiments, therefore, meet with frequent disappointment, and long and tedious preparations are wasted. In this connection it is to be remembered that the experimenter is at present faced by long odds, for the chances at the start are 10 to 1 or more against his seeing, in any given animal, a period of pure flutter of sufficient duration to enable him to complete the necessary observations.

in all directions and at more or less uniform rates; as the intra-auricular band (Fig. 15, Point *e*) lies closer to the inferior vena cava than does the cephalic end of the tænia (Fig. 15, point *c*), so the propagated waves reach the former point before they reach the latter. The waves of excitation proceed up the tænia terminalis throughout its whole length, but when they turn the corner and course to the left of the superior cava they meet the corresponding waves, as these proceed through the tissue of the left auricle to the same region of the heart. The meeting point is not far

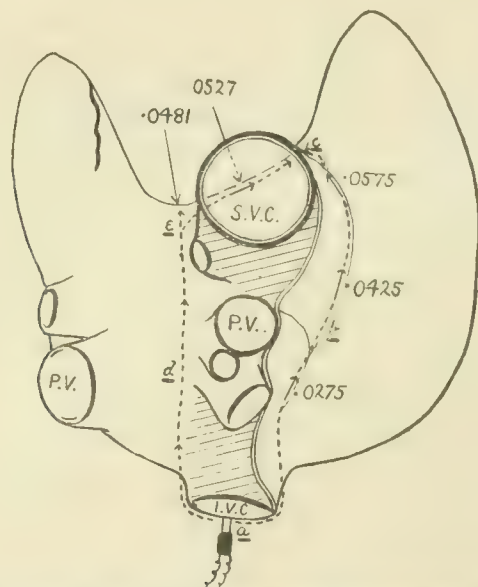


Fig. 15. Dog KW. (Records 19-23.) An accurate and natural size drawing of the auricle of dog KW, to illustrate the path taken by the excitation waves propagated artificially (at *a*) from the inferior vena cava. Five points on the auricle were examined and the corresponding readings are given. The readings represent the intervals between the stimulus and the corresponding excitation wave. The dotted line and arrows represent the path pursued. S.V.C.= superior vena cava and I.V.C.= inferior vena cava. P. V.= left and right pulmonary veins.

from the base of the right appendix, as Fig. 15 shows, and the direction of the excitation wave in the band is from left to right. In this region the wave follows a path directly opposed to that prevailing in the form of flutter, in which the wave is first found issuing from the inferior cava. The course of the wave during inferior vena caval stimulation is precisely what would be expected of it, having regard to the anatomy of the auricle and the relation of its parts.* The further to the left the point stimulated is chosen,

* The experiment cited is one of three, in all of which the results were in essential agreement. The point of meeting may be a little further to the left, that is to say, a little way down the band; but in all it is quite clear that point *e* is activated as early or earlier than point *c*.

the more certainly will the wave traverse the band from left to right and the earlier will it reach the left appendix, a point reached at a very late stage during the progress of flutter.

Now, if the natural course of the wave during inferior caval stimulation is from cava directly to band and through this from left to right, it follows that, when we discover during flutter that the excitation waves proceed from the inferior caval region and pass up the tænia and around the superior cava to proceed from right to left through the whole length of the band, the direct path from inferior cava to band is already closed. It is closed because the tissue which composes this path is in the refractory state, the wave having recently traversed it. It is also to be observed that the form of the auricular complex in lead *II*, when the inferior cava is stimulated, is not the same as that which prevails in flutter of the form described. We are able to publish a curve (Fig. 32) from *Dog JT*, in which this is shown. Fig. 32 should be compared with the remaining figures taken from the same animal during the period of flutter.

In the light of these experiments we may conclude more positively that flutter of the auricle is due to the establishment of circus movement. This conclusion is found to be compatible with, nay explains and is therefore supported by, many considerations which will be discussed subsequently.

OBSERVATION 5. (*Dog KD*. WEIGHT 24·7 KILOGRAMMES.)

If circus movements become established in the right auricle it may naturally be asked why they may not appear in the left auricle. It seems probable that they do; but because this auricle lies deeper buried, and especially because of its relation to the pericardial reflections, such circuits must remain difficult to prove. From time to time flutter of the auricle is seen in which the readings are arranged differently from those already instanced. A not uncommon variety of flutter is exemplified by the present experiment. The flutter followed rhythmic stimulation of the *S. A. N.* region at a rate of 379 per minute and it lasted 74 minutes and 40 seconds. Many electrograms* were obtained from surface points in this animal, but some were unsatisfactory, for the following reasons. Firstly, the auricular complexes in lead *II* were small and difficult to define, and in a number of plates the standard point of measurement could not be identified with certainty; such curves have been excluded. In the second place, when this experiment was undertaken, we were under the impression that flutter consists always of a movement circulating about the largest veins. A number of electrograms taken from the circumference of the superior cava, to which especial attention was directed as a consequence of this view, proved of no special value. One of the chief difficulties in mapping out the

* The term electrogram is used to distinguish a curve from a direct lead, as opposed to the term electrocardiogram, which is applied to leads across the whole heart (lead *II*).

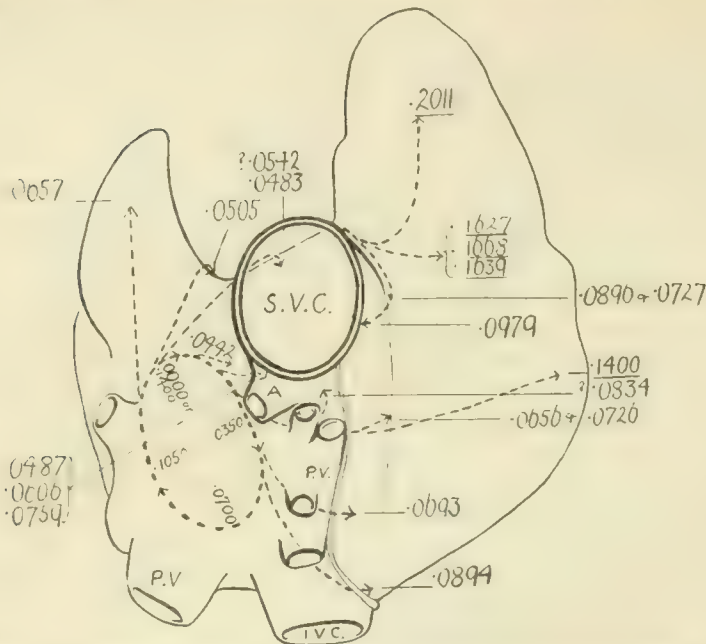


Fig. 16. *Dog KD.* (Records 29a-48.) An accurate outline of the auricle of *Dog KD*, reproduced at natural size. The readings have been reduced to a new common zero (see Table VI and Fig. 18). The heavy broken circle represents the supposed track of the circus movement around the left auriculo-ventricular orifice. The readings inside this cycle are hypothetical. The arrows are drawn in directions which are compatible with the path of the excitation wave, as ascertained by the directions of the deflections in direct leads, and with the readings obtained from the surface of the heart. *S. V. C.* = superior and *I. V. C.* = inferior vena cava. *P. V.* = left and right pulmonary veins. *A* = azygos vein.

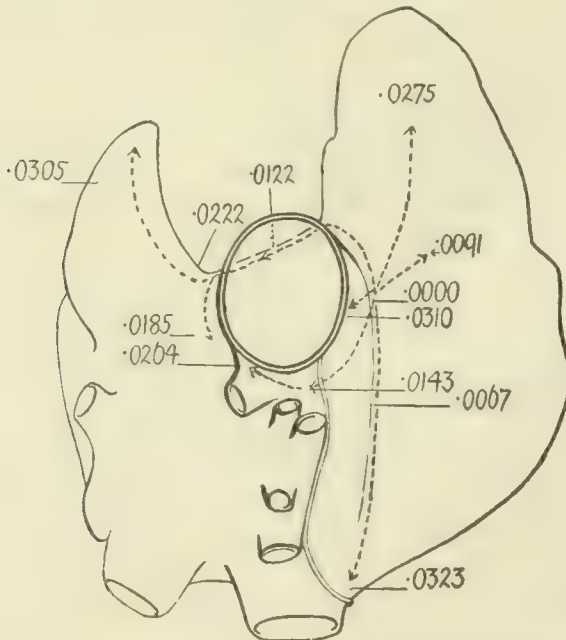


Fig. 17. *Dog KD.* (Records 49-60.) A similar outline showing the readings and the direction in which the excitation moved while the heart beat naturally. The readings have been reduced to a new zero, namely, the reading obtained from the *S. A. N.* region.

path of the excitation wave in flutter is that there is little clue to the path beforehand, and it is not until the curves are measured and the values are calculated that the general direction of flow becomes clear. The only clue during the progress of the experiment is obtained by watching the movements of the string shadow on the camera as the plates are taken: from this the direction of the intrinsic deflections may often be gauged, and it may be recognised that the excitation wave flows to the *Z* or *C* contact first.* But when, as in the present experiment, the excitation wave flows in unexpected directions, the contacts are often placed on an unsuitable point or in an unsuitable plane. In the last case curves showing many phases are obtained and the intrinsic deflection in these circumstances cannot be identified with the same certainty: a number of records taken from this animal belonged to this class: many of them were of no value, others gave readings (marked ? in Fig. 16) which were of questionable value. But as the flutter in this animal lasted for an unusually long period, we were able to obtain an unusual number of readings, and these have been reduced to a new zero and are charted in Fig. 16. The actual relations to the auricular complex is shown in Fig. 18.

Readings from the line of the tænia. From the upper end of the tænia (*S. A. N.* region) two readings of 0.0727 and 0.0896† were obtained. From the mid-caval region readings of 0.0656 and 0.0726 were obtained, and from the upper and lower parts of the inferior cava the readings were 0.0693 and 0.0894. The course of the excitation wave differs from that witnessed in previous experiments, in that it reaches the whole line of tænia almost simultaneously: the form of the electrograms agrees with this finding, for contacts placed in the line of the tænia showed small or multiple deflections.

Readings from the left auricle and dorsal wall of the superior cava. The readings from the left wall of the superior cava (0.0483 and 0.0542) and band (0.0505) were exceptionally low, and the curves showed the wave to be travelling from left to right. In preparing the animal for this experiment the pleura had been incised along the right and dorsal wall of the cava, and the vessel had been dissected away from its bed of connective tissue, so that the posterior wall of the vessel might be reached. In the deepest part of this dissection we had hoped to obtain a reading from the left auricle, behind the superior cava.‡ The figure 0.0442, which is charted in Fig. 16, corresponds to a curve taken from this region, but, as subsequently transpired, the contact

* Frequently, however, this clue is not obtainable, and it is not until the observations of this experiment. Further, small deflections may be observed, but it is necessary, in the absence of any other clue, to take the plates to be taken as rapidly as possible. There is no time to develop and examine each plate as it is exposed.

† The second reading, 0.0896, is from the inferior cava, and is not a reading from this experiment is the reading of the intrinsic deflection from the *A-V* groove separating right auricle and ventricle (0.0000, or 0.1400 as charted).

‡ With the object of completing the readings and filling up the gap left in this part of the circus in previous experiments.

lay a centimetre or a little less from the actual muscle. Through the same incision in the pleura, three readings (0.0487, 0.0606 and 0.0759) were obtained from the dorsal wall of the cava, at varying distances beyond the azygos vein (marked *A* in the chart). These three readings are not in very close agreement, we presume because the level of the lead was altered, but one of them (0.0487) is conspicuously early. All the curves from this region, that giving the reading 0.0442 and those taken from the dorsal wall of the cava, showed by the direction of the intrinsic deflections that the wave passed from above downwards in the auricle as it stands in our chart.* A lead from the left appendix gave an exceptionally low reading in the light of our previous experience; the wave was shown to proceed from its base to its tip.

Right auricle. From the base of the right appendix (0.1627, etc.) and from the *A-V* groove (0.1400) four curves were obtained; these all showed the wave to be proceeding from left to right, as indicated by the broken arrows. The curve from the tip of the appendix (0.2011) indicated the usual movement from its base to its tip. But in respect to the actual readings from these regions of the auricle a doubt exists. It will be obvious from a consideration of Fig. 18 that in the original measurement of a curve, any given intrinsic deflection may be expressed as a + or a - quantity, for it lies after one auricular summit and before the corresponding summit of the next cycle, in lead *II*. It is a matter of indifference in which way it is expressed, providing that the cycle to which it belongs is recognised. Such recognition is only possible when the readings from a line of points on the muscle are arranged in an orderly sequence, as is the case in Figs. 7 and 11. But where the sequence is not so plain there is no such certainty. In the examples of flutter which have been described previously the complete range of readings lies within the length of the single auricular cycle; but we have no guarantee that this is always the case,† and the present experiment forms such an exception. The readings for the right auricle now under consideration may be taken at the alternative figures given in Table VI. The readings at first accepted were the lower readings in each case, namely, 0.0611, 0.0227, 0.0268, 0.0000 and 0.0239; but no one of these readings, with the exception of that from the tip of the appendix, is compatible with the remaining indications of the direction in which the excitation wave moved. To render our readings compatible with the direction of the intrinsic deflections, etc., we are compelled to move them into the succeeding auricular cycle, raising each value by 0.1400‡ of a second. It is recognised that this procedure is an arbitrary one, but without it we can explain the readings and the directions of the deflections, neither on the basis of a circus movement nor in any other fashion.

* As the auricles stand in our charts the ventral surface is towards the right.

† In other words, it is possible that one auricular cycle may still be in progress, in the further most points of muscle, for some time after the succeeding cycle has started.

‡ The length of a full auricular cycle.

TABLE VI.

*Readings in a long period of auricular flutter.**From 10-1 K.D.*

Points investigated	Record No.	Minutes after onset of flutter	Summation of <i>P</i> to intrinsic	Intrinsic readings reduced to a new zero	Auricular rate.
<i>S. V. C.</i> (posterior wall)	29a	1.1	+0.173	0.739	437
<i>S. V. C.</i> (left wall)	29b	1.7	+0.144	0.712	435
<i>S. V. C.</i> (right wall)	30a	2.5	+0.248	0.834	432
<i>S. V. C.</i> (posterior wall)	31a	6.1	+0.229	0.487	425
<i>S. V. C.</i> (left wall)	31b	7.0	+0.193	0.483	424
Inferior vena cava (upper)	33a	16.2	+0.107	0.693	407
Tip of right appendix	33b	17.0	+0.1425 +0.0025	0.2011 0.611	404
Band	34a	25.3	+0.081	0.505	420
Left appendix	34b	26.9	+0.071	0.657	416
<i>S. V. C.</i> (posterior wall)	35	31.3	+0.020	0.606	414
<i>S. A. N.</i> region	38	37.5	+0.310	0.896	415
Left auricular lead	39	40.3	+0.144	0.442	410
<i>S. A. N.</i> region	40	44.0	+0.141	0.727	400
Base of right appendix	41	47.1	+0.1041 +0.0359	0.1627 0.0227	402
<i>S. V. C.</i> (up the vein)	42	50.3	+0.393	0.979	401
Mid-cava	43	53.9	+0.140	0.726	404
Inferior vena cava (lower)	44	54.7	+0.308	0.894	396
Mid-cava	45	57.1	+0.070	0.656	404
Base of right appendix	46	59.5	+0.1082 +0.0318	0.1668 0.0268	404
<i>A-V</i> groove (right)	47	67.3	+0.0814 +0.0586	0.1400 0.0000	404
Base of right appendix	48	72.7	+0.1053 +0.0347	0.1639 0.0239	397

Assuming that the readings as they stand in Fig. 16 to be accurate, we may explain them by supposing the circus movement to take place in a clockwise direction around the left auriculo-ventricular orifice as indicated in the figure. The figures placed *inside* the broken ring are, of course, hypothetical.

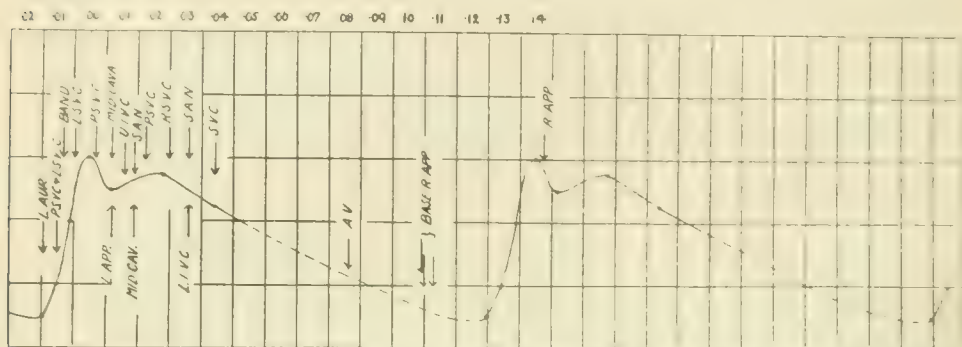


Fig. 18. *Dog KD.* (Record 18-18.) A chart constructed in the same fashion as Fig. 4, to show the relation of the auricular leads to the auricular complex in lead *II* during a long period of flutter. The broken line of the curve represents that part of it of which the values are uncertain. *L. A.U.*=point on the lateral side of the superior cava (Fig. 16 reading 0.0442). *P.S.V.C.*= posterior or dorsal wall of the superior cava; this lies to the left in Fig. 16. *L.S.V.C.*= left wall of superior cava; this lies above in Fig. 16. *U.I.V.V.* and *L.I.V.C.* leads from the inferior vena cava (readings 0.0693 and 0.0894 respectively in Fig. 16) *R.S.V.C.*= right surface of superior cava; this lies below in Fig. 16. *A.V.* a point near the *A-V* groove, marked by the reading 0.1400 in Fig. 16. Ordinates=one-tenth millivolt. Abscissae=0.01 of a second.

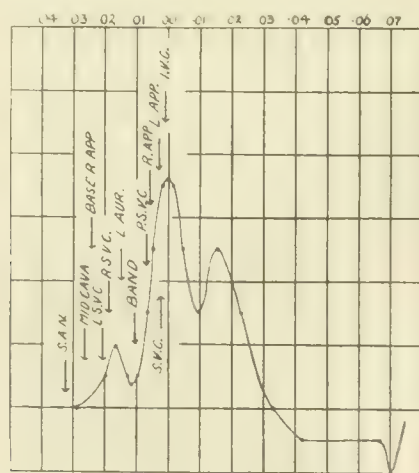


Fig. 19. *Dog KD.* (Record 49-60.) A chart of the same kind, constructed from readings taken while the heart beat normally. The lettering on this chart is similar to that of Fig. 18. Ordinates=one-tenth millivolt. Abscissae=0.01 of a second.

This same auricle passed into flutter for a period of 29 minutes, during which the four sides of the superior cava, the band, the mid-caval region and the right appendix were investigated. The curve from lead *II* was

similar to that shown during the period of flutter previously described, and the direct readings from the muscle were in close agreement with those already discussed.

To sum up, the observations on this dog seem to us to be compatible with a circuit movement in the left auricle; but while this appears to be a reasonable explanation, we fully realise that the experiment does not prove that such a circuit was present; our evidence does not appear to *prove*. We describe it because it represents a form of flutter differing materially from that previously recorded in this paper; because it illustrates some of the difficulties with which investigations into the nature of auricular flutter are beset; and, lastly, because the details may possibly prove of value in future studies of similar mechanisms.

The path taken by the normal excitation wave in this animal is shown in Fig. 17; the corresponding chart, relating the intrinsic deflections to *P* in lead *II*, is shown in Fig. 19. These two diagrams are in agreement with similar diagrams illustrating the normal heart rhythm in other animals.

HISTORICAL.

In our work upon auricular flutter we have been guided to our conclusion by the observations of other writers. We have been guided especially by an observation recorded by the late Dr. G. A. Mines. Mines experimented on ring preparations cut from the auricles of large rays. On page 383 of his paper⁶ he says:—"In such preparations a single stimulus applied to any point in the ring starts a wave in each direction. The waves meet on the opposite side of the ring and die out. But by the application of several stimuli in succession it is sometimes possible to start a wave in one direction while the tissue on the other side of the point stimulated is still refractory. Such a wave runs round the ring sufficiently slowly for the refractory phase to have passed off in each part of the ring when the wave approaches it. Thus the wave circulates and may continue to do so for fifty revolutions or more." This paragraph is taken from an addendum to Mines' main paper. In the main paper he discusses "circulating rhythm" which he had demonstrated in a less impressive fashion in experiments of a different kind (to which we shall refer presently), and applies it in explanation "of the important and interesting condition of delirium cordis or fibrillar contractions." In the same paper Mines refers to the experiments of A. G. Mayer,⁷ who produced circulating rhythms in rings cut from the bell of Medusa.* Mayer showed that by stimulating he could induce the wave to travel through the ring in one direction and to continue around the ring

* It is an interesting fact that the bell of Medusa should again form the starting point of observations throwing light upon human pathology. As is well known, the experiments of Romanes on jelly-fish led to Gaskell's classical experiments on conduction in the cold-blooded vertebrate heart.

as a continuous circulating wave for hours or days. In a later paper,⁹ published after his death, Mines repeats his former statements and describes further experiments. In rings of the auricular tissue of the dogfish and in rings cut from the hearts of dogs, he was able to induce similar circus movements by appropriate stimulation. Mines believed that such circulating rhythms were the cause of the condition known as fibrillation. Independently of Mines, Garrey¹ was working on similar lines and came to much the same conclusions. Garrey's experiments were undertaken on rings cut from the base of the ventricles of large turtles; in this he produces "circus contractions," and for reasons which he states in his paper he sees in these "circus contractions," determined by the presence of blocks, the essential phenomenon of fibrillation. The same idea has been advocated by Levy², and by McWilliam,⁶ and had indeed been put forward in less distinct forms by McWilliam⁵ in 1887, and by Porter¹⁰ in 1894. In a recent publication³ one of us has discussed at some length this view of the mechanism of fibrillation, and has pointed out that fibrillation cannot be explained on the basis of a simple circus movement.* Now, this is not the place to discuss theories of the underlying mechanism of fibrillation,† but, since the nature of flutter and fibrillation are so clearly interwoven, it is not permissible to omit these references to the writings of others at the present stage.

The point which at present concerns us is this: it was recognised that although the hypothesis of a solitary circus movement is inapplicable as an explanation of fibrillation,‡ it might be applicable to auricular flutter.§

In this connection we desire to record our belief that had Mines or Garrey been more familiar with auricular flutter, as it is seen in the human subject, they could scarcely have avoided arriving at the same preliminary hypothesis. Mines indeed came very close to it. In his articles he described a preparation of the tortoise heart, in which a ring of tissue is cut vertically from the whole heart, so that its upper quadrants consist of auricle and the lower quadrants of ventricle. This ring preparation includes two separate segments of the *A-V* junction. Mines showed that a wave may be induced to travel around this ring in one direction, passing from ventricle to auricle and back from auricle to ventricle, and so on.|| Mines believed that if such a reciprocating mechanism were possible in the cold-blooded heart, it might

* Mines' paper appeared in 1913. It is to be regretted that at the time when I wrote this review³ I had not fully appreciated that both Mines and Garrey were not postulating a simple circus movement, but were fully alive to the possible presence of many such circuits in one mass of muscle, circuits subject to change from time to time.—(T.L.)

† That will be undertaken at a later stage.

‡ As stated in a preceding footnote, such a theory was not actually put forward.

§ And a statement to this effect was made in the review to which reference has been made.

|| We recall a number of isolated observations in past writings in which a reciprocal action of auricle and ventricle has been postulated, to account for short series of alternating contractions of the auricle and ventricle. In the tortoise heart the conducting tissue of the junction is not confined to one region of the ring.

also be possible in the mammalian heart;* and on this ground he attempts to explain "some cases of paroxysmal tachycardia as observed clinically." But our clinical observations and experimental work on the mammalian heart are distinctly opposed to such a view.

The conception with which we started our observations was a different one, for it confined the simple circuit to the auricle and limited the explanation to a single disorder of the heart beat, namely, auricular flutter of shorter or longer duration. This conception was based upon our recognising that the mouths of the veins and the muscle surrounding the *A-V* orifices constitute natural rings in the intact heart, in which circus movements might therefore be propagated. The conception was an outgrowth of those of other writers, and especially it was an outgrowth of the simple and dramatic experiments which Mines describes. The present article examines our preliminary hypothesis in the light of direct observations, and brings us to the conclusion that it is sound.

The detailed theory of circus movement in rings of tissue such as Mines and Garrey used, and in the intact auricle, may be conveniently deferred until further observations relating to flutter are recorded in subsequent articles.

NOTE.—One of us² has put forward an alternative hypothesis, that flutter may consist of simple paroxysmal tachycardia, arising in the pacemaker. This requires no further discussion, as it is quite incompatible with the observations recorded in the present paper.

SUMMARY OF OBSERVATIONS AND CONCLUSIONS.

A condition of the auricle may be provoked experimentally by means of rhythmic induction shocks at different rates, which is identical with auricular flutter as this is observed in the human subject. It is recognised by the following peculiarities, which it holds in common with the clinical condition :—

(a) The rate of the auricular movements is very rapid. In the dog the limits are 345-580 per minute.

(b) The movement of the auricle shows a high grade of regularity. In the dog, when the cycles are measured in direct leads from the auricle, the length of the cycles usually varies about the average value by no more than + or — 0.001 to 0.003 of a second, exceptionally by 0.007 of a second.

(c) The auricular complexes are contiguous and are uniform in outline.

It seems desirable that the unqualified term "auricular flutter" should be confined to mechanisms of this kind.

* Mines cites Stanley Kent's anatomical researches to this end. In this we believe him to have been at fault, as no evidence has yet been brought forward there is much to the contrary to show that a path of conduction between the auricle and ventricle in the mammalian heart other than the *A-V* bundle exists.

The following observations apply to auricular flutter as it is seen in experiment:—

(1) The excitation wave passes through the auricular tissue in a perfectly regular sequence from cycle to cycle over considerable stretches of curve. It is suggested that this phenomenon of ordered activity is a constant association of rhythms displaying high grades of regularity.

(2) In a number of experiments it has been shown that throughout the whole auricular cycle some part of the auricular tissue is becoming activated. There is no true diastole, using this term in the sense that at a given instant all portions of the auricular muscle are at rest. The contiguity of the auricular complexes in limb leads is fully explained by this continual activation of some new portion of the muscle.

(3) The observed rates of conduction in the muscle of the auricle in which auricular flutter prevails are slower (often considerably) than the rates observed when the normal rhythm prevails.

(4) In a number of auricles the direction taken by the excitation wave during periods of flutter has been studied. The path taken by the wave is judged from the directions of the deflections in direct leads and by timing the excitation wave as it passes chosen points. These data are in agreement. The direction taken by the wave, once flutter is definitely established, is not governed by the point at which the auricle has been stimulated. The condition flutter results from stimulation, but becomes independent of this for its continuance. In several animals the course of the surface wave has been mapped out almost in its entirety, and these data point to the existence of a wave circulating continuously through the body of the auricle.

(5) The course taken by the wave, when that course is distinctly displayed, is around the orifice of the superior vena cava, and surrounds a certain portion of the tissue lying on the inferior caval side of the superior orifice; in some instances it surrounds the mouths of both the superior and the inferior vena cava. The movement may be in either direction,* namely, down or up the *tænia terminalis*. It is to be concluded that this *central wave* constitutes a continuous circus movement in a natural ring or cylinder of muscle existing in the intact heart. The presence of such a circulatory mechanism would explain long continued flutter in the human subject and the high rate of auricular beating. It would also explain a fact, which has been pointed out previously³ but which has received as yet no adequate explanation, namely, the close family resemblance of auricular flutter curves taken from different patients.

* Clockwise or anticlockwise.

(6) Those portions of the auricle, such as the appendages, and the sleeves of muscle on the great veins, are supplied by excitation waves flowing centritugally from the body of the auricle. These *centrifugal waves*, so it is concluded, are thrown off from the central path, as the central wave revolves in the latter. Thus, in different experiments the *direction* of the wave in these outlying parts is in large measure constant and independent of the direction of movement in the central path: but the times at which these waves travel through the outlying parts is controlled by the central movement.

(7) During the progress of long-continued experimental flutter there are in some experiments indications that, although the general path travelled remains constant, slight variations from that path may take place. Strictly speaking, when such variations occur the auricular flutter is impure.

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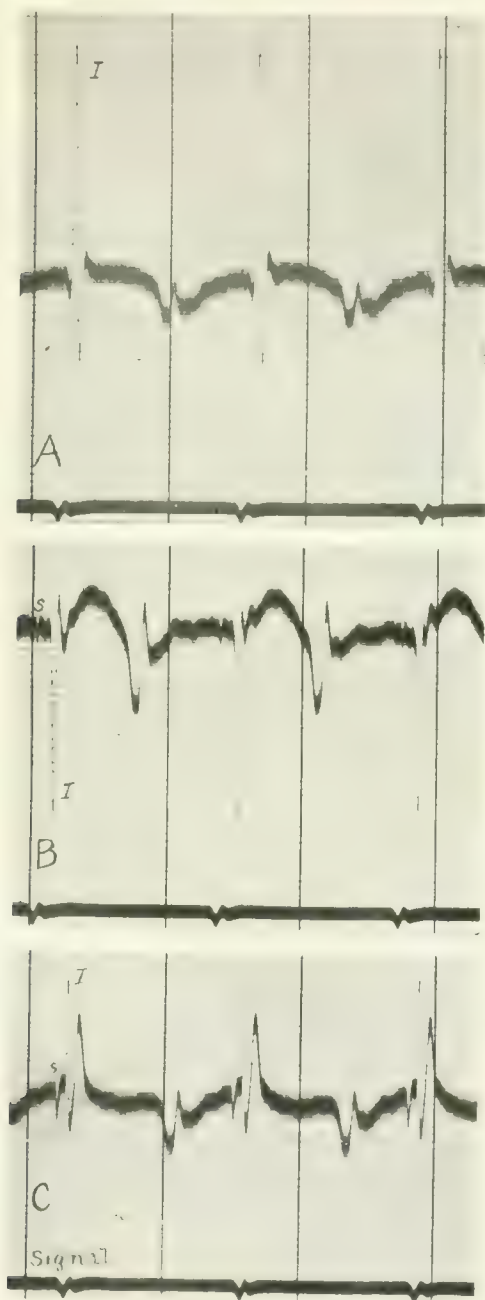


FIG. 20. Dog K II. (Records 1, 2 and 3.) The auricle is responding to rhythmic stimulation (see signal below each curve) applied in line with (A and B) or at right angles to (C), a pair of contacts resting on the surface of the auricle (see Fig. 1). In A the point stimulated was on the side of the Z contact, and a tall upward intrinsic deflection *I* is recorded. In B the point stimulated was on the side of the C contact, and a deep downward movement is registered. In C the excitation wave strikes both contacts almost at the same moment, and several deflections of lesser amplitude result. Time lines in fifths of a second. Ordinates, 1 centimetre = 3 millivolts.

In taking the following curves the standard of the electrocardiograms has been 1 centimetre = 1 millivolt, and of the electrometers it has been 3 to 5 millimetres = 3 millivolts. As little attention could be given to the excursion of the string during the progress of the experiments, the values are only known approximately for the later curves of each experiment. In all curves the three lines mark fifths of a second. Some of the records have been reduced a little. In the original the horizontal lines stand 1 millimetre apart.

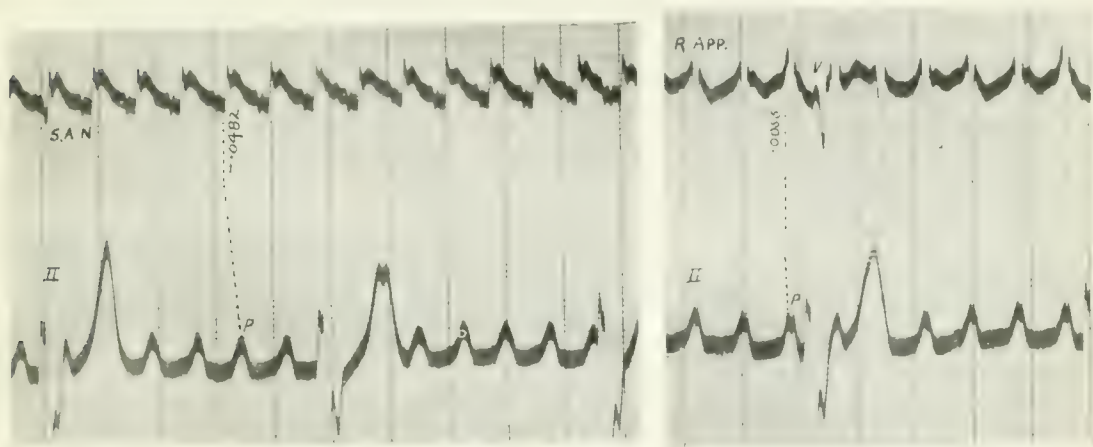


Fig. 21. *Dog J.P. (Record 9.)* A curve taken by a direct lead from the S. A. N. region (Z contact below, C contact on superior cava; see Fig. 2) and electrocardiogram from lead II. Complete A-V heart-block had been produced in this animal by clamping the bundle, and the right division of the bundle was also involved. The intrinsic deflection starts in the average 0.0482 of a second before the summit of the auricular complex (P).

Fig. 22. *Dog J.P. (Record 10.)* A curve taken by a direct lead from the right appendix (Z contact towards the tip of the appendix) taken simultaneously with a curve from lead II. The intrinsic deflections are of great amplitude and downwardly directed. They precede the summit of the auricular complexes in the average by 0.0055 of a second. The beat of the ventricle leaves its impression on the direct lead (see *v*).

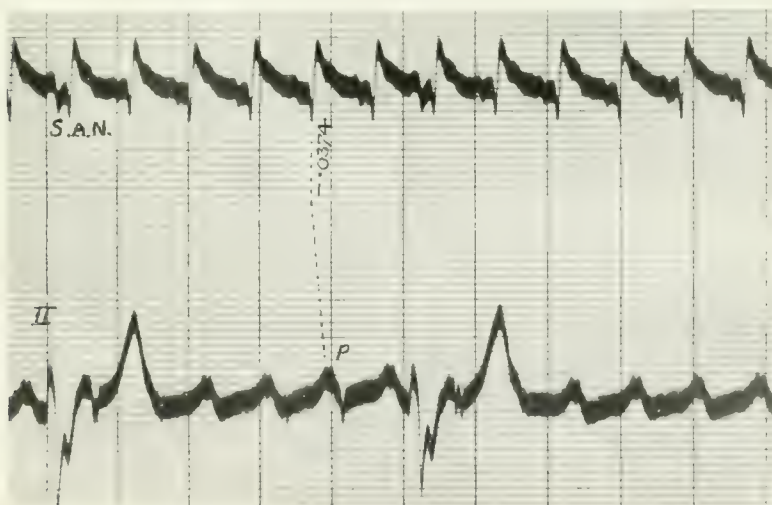


Fig. 23. *Dog J.P. (Record 17.)* A curve taken by a direct lead from the S. A. N. region (Z contact below, C contact on superior cava; see Fig. 3), taken simultaneously with a curve from lead II, during the second after-effect. The intrinsic deflections are upright and precede the summit of the auricular complexes by an average of 0.0374 of a second. Compare with Fig. 21. The standard of lead I I begins to fall a little from this curve onwards.

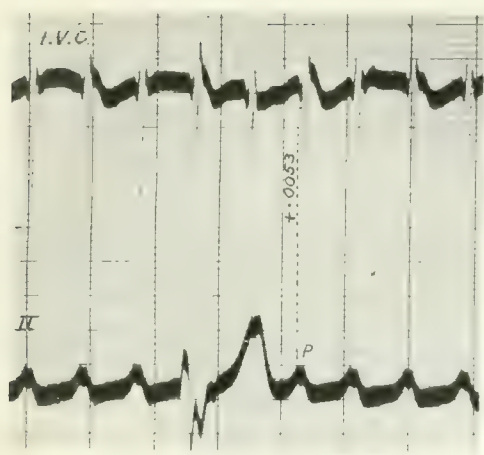


Fig. 24. *Dog I.P.* (Record 15.) A curve taken by a direct lead from the inferior vena cava (Z contact below) and a curve from lead *II*. The deflections are downwardly directed and follow the auricular complexes by an average of 0.0053 of a second.

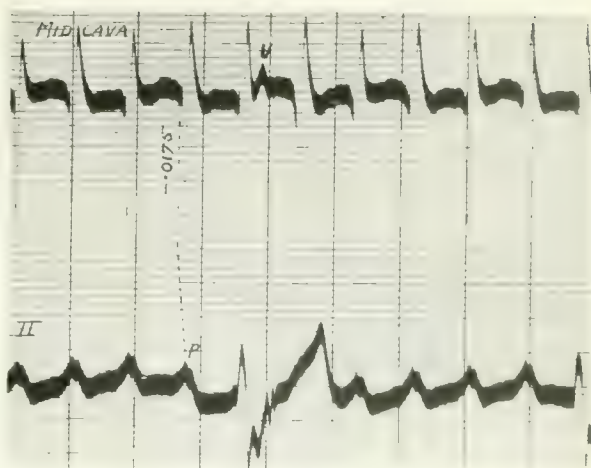


Fig. 25. *Dog I.P.* (Record 18.) A curve taken by a direct lead from the mid-caval region (Z contact below) and a curve from lead *II*. The deflections are downwardly directed and precede the summit of the auricular complexes by an average of 0.0175 of a second.

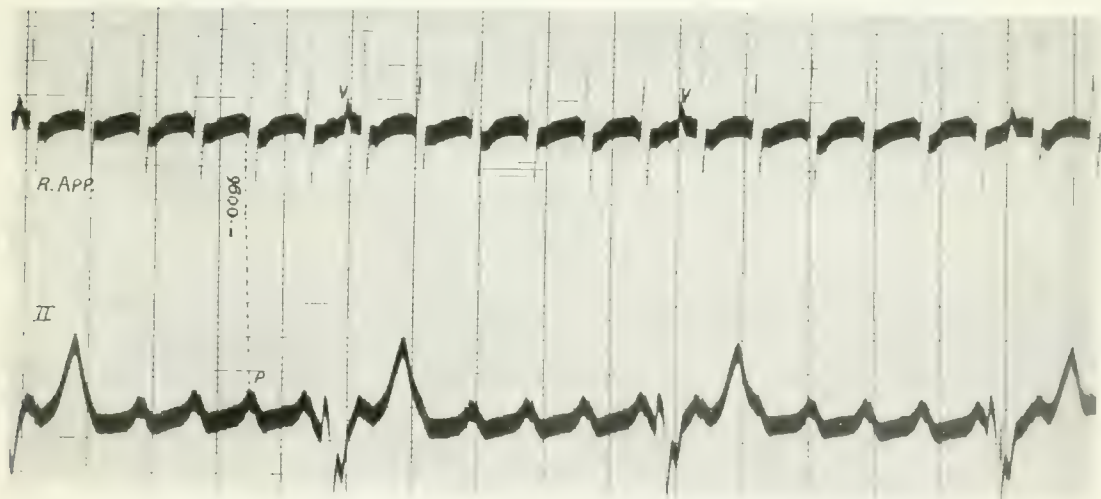


Fig. 26. *Dog I.P.* (Record 16.) A curve taken by a direct lead from the tip of the right appendix (Z contact below) and a curve from lead *II*. The intrinsic deflections, which are upright, precede the auricular summit by an average of 0.0096 of a second.

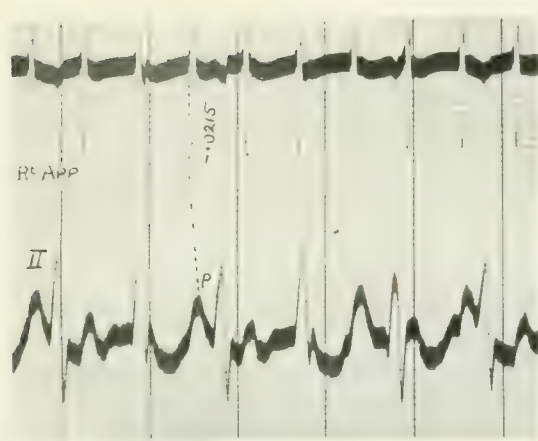


Fig. 27. *Dog J.T.* (Record 14.) An electrogram from the tip of the right appendix (Z contact above, see Fig. 7) and an electrocardiogram from lead II. The intrinsic deflections are downwardly directed and precede the auricular summits by an average interval of 0.0215 of a second.

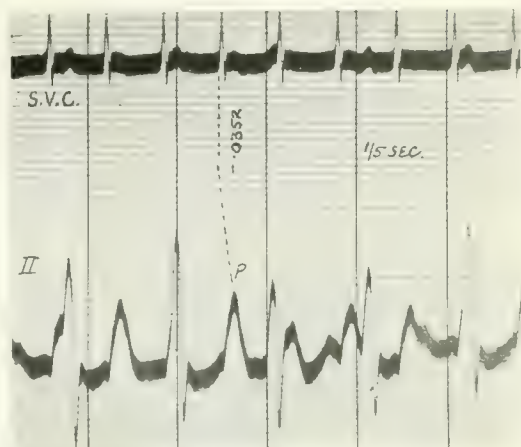


Fig. 28. *Dog J.T.* (Record 17.) An electrogram from the superior vena cava (Z contact below) and a curve from lead II. The intrinsic deflections are upright and precede the auricular summits by an average interval of 0.0352 of a second.

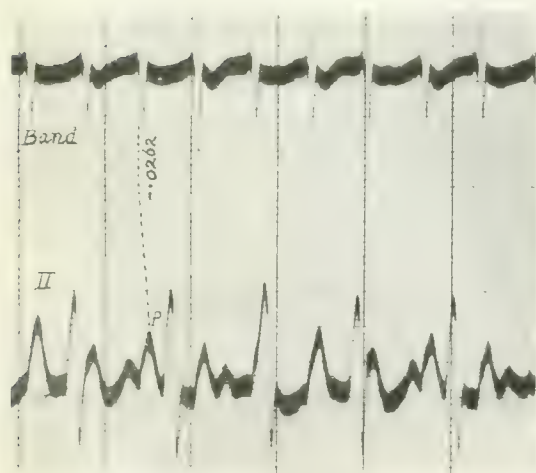


Fig. 29. *Dog J.T.* (Record 18.) An electrogram from the intra-auricular band (Z contact to the right) and a curve from lead II. The intrinsic deflections are upright and precede the auricular summits by an average interval of 0.0262 of a second.

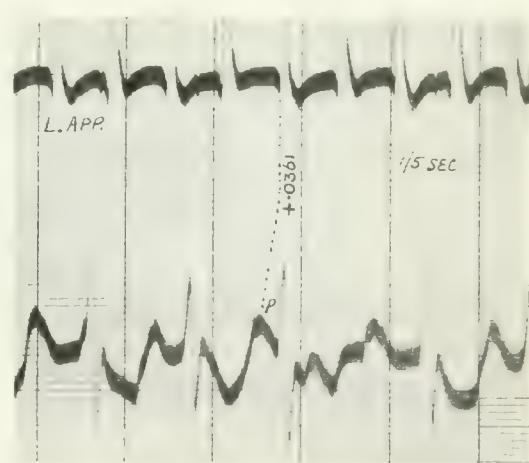


Fig. 30. *Dog J.T.* (Record 19.) An electrogram from the left appendix (Z contact below) and a curve from lead II. In moving the heart to reach the tip of the appendix the outline of the auricular complexes has been altered a little. The intrinsic deflections are upright and follow the auricular summits by an average interval of 0.0361 of a second.

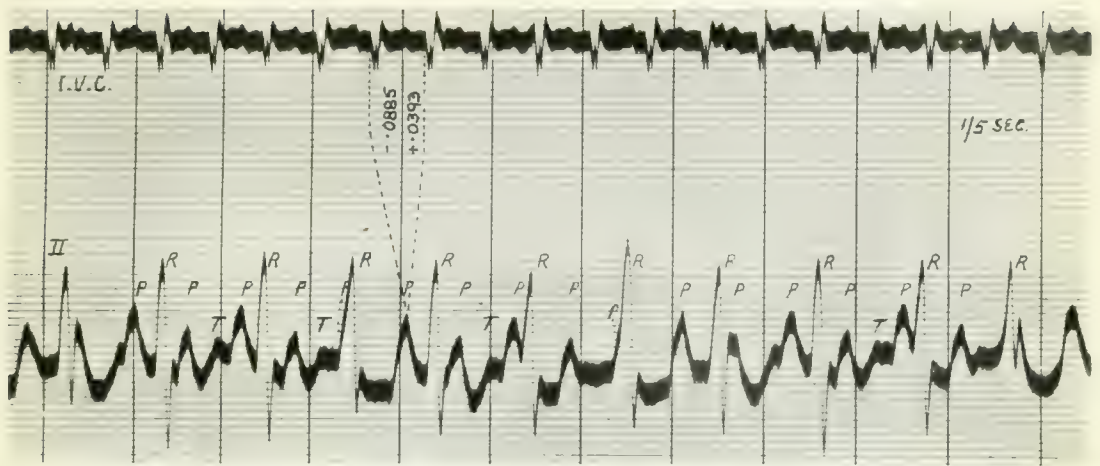


Fig. 31. *Dog JT.* (Record 15.) An electrogram from the interior vena cava—Z contact above—and a curve from lead II. The intrinsic deflections are not conspicuous, but the first prominent deflection precedes the auricular summit by an average interval of 0.0885 of a second; the next deflection succeeds the summit by an average interval of 0.0393 of a second.

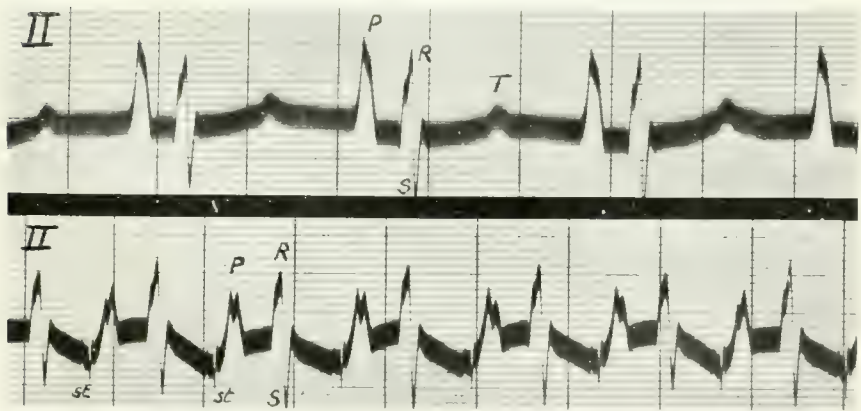


Fig. 32. *Dog JT.* (Record 32.) Two separate electrocardiograms from lead II. The top curve shows the normal rhythm. The bottom curve shows the form of the auricular complex while the heart responds to rhythmic stimulation of the inferior vena cava. The current of stimulation is seen in the curve as a sharp downward deflection *st*. (For comparison with the remaining curves from this animal see Figs. 27-31.)

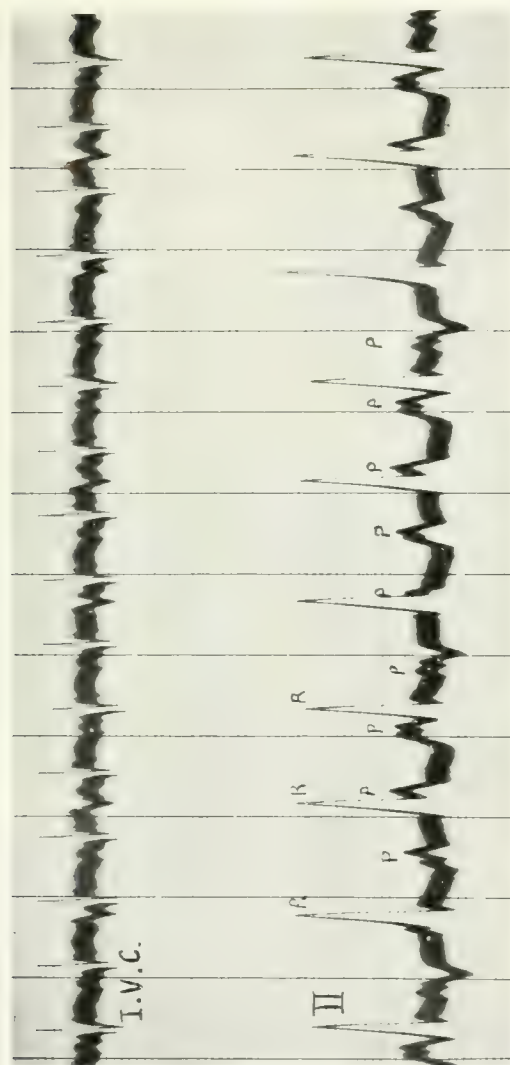


FIG. 33. *Dog KQ.* $R = 32$. An electrogram taken from the site of vein *AVA* (Z contrast below, see Fig. 11), and a curve from lead *II*: taken 9 minutes from the onset of a long period of flutter. The intrinsic deflections are upright and precede the auricular summit by an average interval of 0.1045 (1.2 second).

OBSERVATIONS UPON FLUTTER AND FIBRILLATION.

PART III.—SOME EFFECTS OF RHYTHMIC STIMULATION OF
THE AURICLE.

BY THOMAS LEWIS,* H. S. FEIL AND W. D. STROUD.

(From University College Hospital Medical School.)

IN the previous article we have brought forward evidence to show that in auricular flutter, as there defined, the excitation wave follows a circular and re-entrant path in the body of the auricle. Evidence has also been given to show that the rate at which the excitation wave is conducted during flutter is lower than while the normal heart rhythm prevails. We possess more evidence that this is so than has been stated. This evidence has been derived from the experiments in which the events foreshadowing flutter have been studied. To take the natural order of our observations would have been appropriate had it been possible: but to do so in this instance would have divorced these evidences of lowered conduction from other effects of embarrassed conduction, which it would not have been convenient then to describe. We have therefore delayed a description of these experiments, and incorporate them in the present article.

Relation of conduction to rate of stimulation.

Two pairs of non-polarisable contacts, each pair forming a lead to one string of our galvanometer, are set in line on the auricular tissue;† the auricle is stimulated by means of rhythmic induction shocks, slightly exceeding threshold value, at a point in line with the contacts and some 15 or 20 millimetres away from the nearest of them. The arrangement of contacts to the point of stimulation is illustrated by Fig. 1. The proximal electrodes *P* and distal electrodes *D* are set so that the *Z* contact of each lies towards the point of stimulation. As a rule the four contacts were laid on the body of the right auricle and the tip of the right appendix, or a point

* Working on behalf of the Medical Research Council.

† The centre of the surface of the right auricle was used in all experiments except one, namely, KU (Records 28 to 35) (see Table).

near it, was stimulated. When the auricle is responding regularly to this rhythmic stimulation, both strings (see Fig. 3) show steep deflections in an upward direction, indicating that the excitation waves strike the *Z* contacts first. The direction travelled is indicated by the unbroken arrow (*a*) in Fig. 1. The contact *Z* of the proximal pair picks up the electrical effect

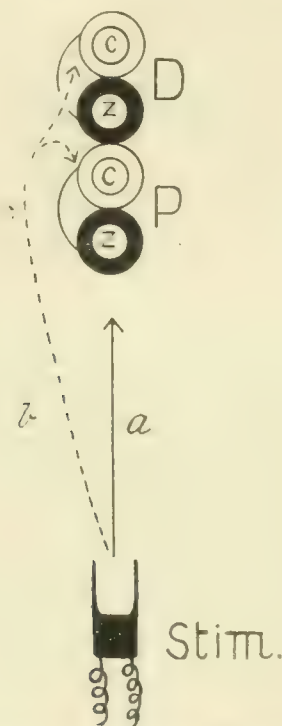


FIG. 1. Four contacts are laid on the auricle in line with a point chosen for rhythmic stimulation. The contacts are joined to the two strings of our galvanometer, so that they form a proximal (*P*) and a distal (*D*) pair, each pair being connected to one string. The connections are so made that the contact of each pair, which lies nearest the point stimulated, yields, when the excitation wave first strikes it, an upright deflection in the curve. It is labelled the *Z* contact (this contact is drawn as a black circle), as opposed to its companion *C* (this contact is drawn as a white circle). The distance between the two *Z* contacts has been uniformly 9 millimetres in the present experiments. The unbroken arrow *a* represents the usual course taken, when the auricle is stimulated in the line of the contacts. The broken path *b* shows a possible path, described at a later point of the text.

before it is picked up by *Z* of the distal pair by a measurable time interval. The distance between the two *Z* contacts has been maintained at 9 millimetres. The rate of conduction is calculated by dividing this distance by the time interval of transmission.*

* The rate of conduction may also be judged from the interval between the stimulus and the intrinsic deflection yielded by the proximal contacts; this interval is an index of the transmission time from the point stimulated to the nearest contact. These intervals have shown similar changes to the interval between contacts, which we tabulated. As the latter are for many reasons the more reliable, we use them alone.

TABLES OF CONDUCTION RATES, ETC.
Dog JQ. (Impure flutter and fibrillation.)*

No. of Record	Heart rate.	Transmission time.	Conduction rate.	Notes.
5	159	0.100	826	
4	183	0.116	776	
1	203	0.100	900	
7	257	0.101	891	
8	268	0.109	826	
6	278	0.103	874	
9	289	0.115	756	
10	312	0.138	652	Slight irregularity in lengths of intervals but not in lengths of cycles.
11	358	0.145	621	Ditto

Faster stimulation rate 429 gave 360 beats per minute and a short after-effect.† The rate of beating was 360 per minute, but it was not possible to gauge the extent of individual responses to the stimulation. Subsequent stimulation at rates of 350-400 per minute gave after-effects:—(1) impure flutter‡ showing excitation waves at an approximate rate of 352 to 385 per minute and lasting 45 seconds to 3 minutes; and (2) auricular fibrillation lasting 25 seconds to 5 minutes.

Dog JR. (Auricular fibrillation and occasional impure flutter.)

No. of Record.	Heart rate.	Transmission time.	Conduction rate.	Notes.
1	233	0.117	769	
4	281	0.107	841	
6	304	0.104	865	
5	310	0.106	849	
7	368	0.108	833	
8	375	0.117	769	Auricular fibrillation, irregularity slight in lengths of intervals.
9	429	0.101	841	
10	436	0.126	714	
12	482	0.139	647	
15	517	0.125	776	

Faster stimulation yielded inco-ordination and a short after-effect. The rate at which beating was 517 per minute. Subsequent stimulation at rates of 400-450 per minute gave the extent to which individual beats were responses to stimulation. Subsequent stimulation gave repeated after-effects, usually consisting of auricular fibrillation lasting from 10 to 40 seconds, occasionally consisting of very impure flutter.

The nature of the after-effects witnessed in the corresponding animal is summarised in these brackets.

† By this term we mean that the deflections of the string were irregular in form and amplitude and that the intervals between corresponding deflections in the two leads were very inconstant.

‡ By impure flutter we mean a condition similar to that described in the last article, but in which the events are not so uniform: it is a condition which will be dealt with fully in a succeeding article.

Dog JS. (Auricular fibrillation and impure flutter.)

No. of Record.	Heart rate.	Transmission time.	Conduction rate.	
3	188	0.149	604	
6	235	0.142	634	
8	273	0.141	638	
10	336	0.148	608	Slight alteration in the heights of deflections but not in the lengths of cycles.
11	372	0.158	570	Slight variation in the heights of deflections and also slight variation in the lengths of cycles.

Faster stimulation (rates 413 and 426) produced inco-ordination and brief after effects; during stimulation the heart still responded to each stimulus, but the variation in the height of deflections and in the length of cycles became greater.

Subsequent stimulation produced after-effects consisting of short and long periods of auricular fibrillation and occasional periods of impure flutter (rate 440 per minute).

Dog JT. (Auricular flutter.)

No. of Record.	Heart rate.	Transmission time.	Conduction rate.	
4	287	0.157	573	
5	311	0.143	629	
8	422	0.143	629	
9	463	0.173	520	
10	541	0.189	476	
11	543	0.214	421	
12	580*	0.273	330	Responding regularly to stimulation.

On stopping this stimulation the auricle continued to beat regularly for $1\frac{1}{2}$ minutes at a high rate (flutter). Stimulation at a rate of 700 subsequently produced: (1) flutter, at a rate of 526 to 460 per minute, and lasting 30 minutes (see Part II, Table IV); (2) flutter, at a rate of 490 per minute and lasting 3 minutes. The first of these periods of flutter is fully described in our previous article.

Dog JV.

No. of Record.	Heart rate.	Transmission time.	Conduction rate.	
3	187	.0140	643	
4	241	.0137	657	
5	245	.0144	625	
6	287	.0150	600	(See Fig. 4.)
7	327	.0154	584	
8	361	.0168 { .0162 } { .0173 }	536	Slight alternation in the heights of deflections in lengths of cycles and intervals (see Fig. 5).

Slightly faster stimulation gave inco-ordination. The rate of stimulation was 417 per minute. The auricle responded at the same rate, but the deflections varied in height and the cycles in their length (see Fig. 6). The ventricles of this animal were accidentally stimulated and fibrillation ensued when the observations had reached this stage.

Dog JV. Auricular fibrillation.

No. of Record.	Heart rate.	Transmission time.	Conduction rate.	
6	158	.0180	500	
7	198	.0173	520	
8	225	.0180	500	
10	237	.0157	573	
12	292	.0153	588	
13	331	.0167	539	
15	372	.0188 { .0180 } { .0197 }	479	Alternation in the heights of deflections and slight in lengths of cycles and in intervals
17	388	.0185 { .0176 } { .0195 }	486	Ditto
18	418	.0194	464	Alternation in height of deflections.

Faster stimulation gave short inco-ordination and after-effects. Thus, stimulation at rates of 422 and 450 per minute gave responses at the same rate, but the deflections varied in height and direction, and the cycles varied in length. Stimulation at a rate of 525 produced short after-effects consisting of auricular fibrillation.

Dog KD. (Auricular flutter.)

No. of Record.	Heart rate.	Transmission time.	Conduction rate.	
61	243	.0125	720	
62	290	.0146	616	
63	332	.0191	471	
64	362	.0184 $\left\{ \begin{array}{l} .0205 \\ .0159 \end{array} \right\}$	489	Alternation in height of deflections and of intervals; and very slight variations in lengths of cycles.
65	393	.0195	462	Slight variations in height of deflections.
66	420	.0219	411	Variations in height of deflections.

Earlier observations on this heart gave short and long after effects consisting of flutter. Thus stimulation at 279 per minute yielded flutter lasting 2 2/3 minutes; the rate of this flutter was 584 per minute. Stimulation at a rate of 350 per minute gave flutter lasting 29 minutes, the rate of flutter being 558 to 511 per minute. Stimulation at a rate of 354 per minute gave flutter lasting nearly 75 minutes; the rate of flutter was 437 to 397 per minute. This flutter is fully described in our previous article (see Part II, Table VI).

*Dog KU. (Auricular flutter.)**

No. of Record.	Heart rate.	Transmission time.	Conduction rate.	
28	243	.0077	1169	
29	259	.0072	1250	
30	298	.0082	1098	
31	340	.0073	1233	Single beat as after-effect.
32	386	.0082	1098	
33	445	.0094	957	
34	456	.0103	874	
35	475	.0110	818	Responding regularly to stimulation.

Stimulation at rate of 554 per minute gave flutter at rate of 488 per minute, and lasting 9 1/2 minutes.

With the single exception of this group of curves, all the experiments of these tables apply to the muscle of the body of the right auricle. In this instance, the muscle was that of the superior vena cava, stimulation being at a point in line with this cava on the body of the right auricle.

*Dog KU. (Auricular flutter.)**

No. of Record.	Heart rate.	Transmission time.	Conduction rate.	
16	259	·0075	1200	
17	278	·0056	1697	
18	303	·0050	1800	(See Fig. 3.)
19	363	·0047	1915	
20	393	·0056	1607	
21	446	·0047	1915	
22	446	·0047	1915	
23	453	·0062	1452	Variation in height of deflections.
24	465	·0082	1098	Variation in height of deflections and very slight in intervals.
25	461	·0115	783	Alternation in height of deflections.

Stimulation at rate of 280 per minute gave almost pure flutter at rate of 487 per minute, and lasting 55 seconds. Repeated several times.

The auricle is found to respond regularly to stimulation at rates lying between the natural rate, prevailing when stimulation is begun, and a maximal rate. This maximal rate is not constant for different animals, but lies usually between 400 and 500 per minute; it may be as low as 350 and as high as 600 per minute. When the maximal rate of response is surpassed certain events happen which will be described later. For the moment we may examine the rates of conduction which prevail at different rates of stimulation, as illustrated by the accompanying tables.

It is the rule to find little or no change in the rate of conduction over a wide range of heart rate. The initial conduction rate, varying widely as it does in different animals (from 500 to 900, exceptionally 2,000 millimetres per second) persists as the auricular rate is raised; it varies little more than is accounted for by error in measurement. But, as the critical rate* is approached, conduction begins to fail: the transmission intervals increase with relative abruptness by some 30, 50 or 100 per cent. The range of rate over which these lengthened transmission intervals may be recognised clearly is usually a comparatively small one. It is small because the lengthened intervals only begin to appear shortly before the critical rate is reached, and because, when that rate is reached, other

* The rate at which after-effects are observed; it may lie below the maximal rate at which the auricle will respond regularly to stimulation.

events often happen which render its measurement less certain. There are, it is true, exceptions to the rule of an abrupt rise of the transmission intervals. These exceptions are to be seen in the observations on *Dogs JT* and *KD*, where the failure of conduction is gradual and extends over a rise of 100 beats per minute; generally speaking, it extends over a rise of 50 beats per minute or less.

Briefly, the power of auricular tissue to conduct is constant for rates up to 300-450 per minute, under the conditions of our experiments: as the rate is raised above these levels, conduction begins to fail; it fails rapidly in most experiments; exceptionally it fails more gradually. The degree of failure witnessed may be expressed in the figures of transmission intervals: these increase by 30-100 per cent. This failure of conduction, consequent upon increased rate of stimulation, leads up, in most animals almost at once, in some animals after a longer preliminary phase, to further events now to be described. These events are of two kinds, namely:—

(1) Changes in the form of the curves obtained while stimulation is still maintained, and (2) after-effects, by which we mean that the auricle continues to beat rapidly for one or more cycles, after stimulation ceases.

1. *Change in the form of curves.*

Associated with the first change in the rate of conduction, there is always a lesser or greater change in the form of the curves. At the lower rates, each beat of the auricle gives a series of deflections of each string: of these deflections the chief, in each case, is a tall spike or *intrinsic* deflection, representing the arrival of the excitation wave beneath the first contact of the pair;* the deflections from cycle to cycle are constant in amplitude and form. When conduction begins to fail it is usual to see a little falling away in the height of the intrinsic deflections, and this diminution is usually exhibited uniformly by the deflections. Less commonly, though by no means infrequently, a condition of alternation is established.

The *alternation* consists of a difference in the amplitude, or a slight difference in the form (or both) of the intrinsic deflections of the curves. Thus, in Fig. 5, although the heights of the upright deflections remain constant in the top curve, the downstrokes are much longer in the second and fourth cycles than in the first, third and fifth. We do not propose to describe in detail the varieties of alternation seen in such curves, but we note their existence. They may occur in the curves of one lead or in both. What is more important is that with few exceptions they are associated either with (a) slight alternation in the lengths of the cycles which display them, (b) slight alternation in the lengths of the corresponding transmission intervals and an increase in the average interval, (c) both. It is

* The evidence for this statement will be found in a recent article.³

the association with lengthened and alternating transmission intervals, especially, which stamps the changes as being due to an alternation in conduction. At the A-V junction alternation in conduction is a very rare phenomenon in the mammalian heart, but has been described (see Lewis and Mathison²). In the mammalian auricle it has not been described previously. In Fig. 5 these alternations in the lengths of cycles and intervals are very small; they amount in fact to no more than a few thousandths of a second in the former and to no more than 1 thousandth of a second in the latter. But the constancy of alternation throughout this curve and in other curves from this and different animals, and its constancy in remeasurement of the same curves, renders it certain that it is not attributable to error in measurement. The observations prove that with the fall in conduction power there is often a variation in conduction through the same strip of muscle at alternate beats.*

Auricular aberration. Very soon, as the rate of stimulation is further raised, there come further changes, and curves of considerable complexity result. To understand such curves—and, as the sequel will show, it is of consequence that we should understand them—very close and often tedious examination of detail is sometimes entailed; the complexity of the curves and the necessity of describing minutiae peculiar to individual curves, forbids the publication of many examples. We choose a single curve which illustrates the main points. It is from the same experiment as Figs. 4 and 5. In Fig. 5 the rate of stimulation was 361 per minute; the rate was raised in several stages to 417 per minute (Fig. 6). The auricle still responded to each stimulus, as a comparison between the curve from the distal lead (*D*) and the signal of stimulation shows, and as was also shown by the immediate cessation of the rapid heart action when stimulation was withdrawn. Now the bottom curve of this figure shows little more than does the top curve of Fig. 5, the cycles are almost regular in incidence. But they are not quite regular; in fact they are not quite so regular as in Fig. 5, which shows alternation. The deflections vary in height and in form. The difference between this curve (Fig. 6, *D*) and that which has been considered (Fig. 5, *P*) lies mainly in the absence of alternation in the present curve and in its replacement by a variation of a more irregular kind and somewhat more advanced type. Of chief interest, however, is the top curve, taken from the proximal lead (*P*). There is, as will at once be seen, a rough correspondence between the incidence of sharp deflection in the upper and lower curves; sufficient correspondence to make it clear that the deflections are due to the same excitation waves in the two curves; but this proximal curve is modified

* It is not necessarily due to a varying rate of conduction through the strip which carries the wave; it may be, and very probably is, due to slight variation in the channel of conduction; but this second explanation presupposes the same fundamental change as the first, namely, depressed conduction in some part of the strip. The essential difference between these possible explanations is that in the first a general depression of conduction at alternate beats has to be conceived, while in the second a local change is assumed.

by additional movements of the string. The sharpest deflections of the curve are downstrokes, starting from the dotted lines which are marked above to indicate the intervals between them. Each of these downstrokes is preceded by a more or less abrupt upstroke, to which the dotted oblique lines are drawn below the curve *P*. Broadly speaking, each auricular complex of this curve consists of *a more or less abrupt upstroke* (usually itself preceded by a little dip), a period of hesitation or of dipping, and *a sharp downward movement* of the string which crosses the base line and then returns to it. It is to the main upstroke and the main downstroke to which we draw special attention. There can be no doubt, we think, that the sharp downstroke marks the arrival of the excitation process beneath one or other of the proximal contacts.* If that is the case, then it represents its first arrival at proximal contact *C* (see Fig. 1). In other words, if we accept this explanation of the curve, in so far as it has been taken, we must admit that the general linear course of the excitation wave across the four contacts, from the point of stimulation, has been departed from, and that the point of entry to the region of the contacts, is now somewhere between the two pairs† (as indicated by the dotted path in Fig. 1). If that was the case, we should expect to find that the interval between the arrival of the excitation wave at the proximal and distal pair of contacts has been reduced. In Fig. 5 it averages 0.0168 of a second. In the present curve it averages 0.0068 of a second; it does not exceed 0.0142, is variable and sometimes sinks almost to zero, and at times goes somewhat beyond to the negative values of 0.0024 and 0.0016 of a second. There is a feature of the curve which strongly supports our interpretation; the more distally the excitation wave enters the gap between proximal contact *C* on the one hand and distal contact *Z* on the other, the more likely is it to flow back over the proximal contacts and produce a pure downstroke. It is noteworthy that the purest downstrokes in this curve, those which start from the lowest level, are all associated with intervals which approach closely to zero. Where the interval approaches 0.0100 of a second, indicating that the excitation wave has entered the gap nearer to *PC* than to *DZ*, the downstroke starts at a much higher level and is preceded by a small but sharp upstroke (see first footnote on this page). There seems, therefore, to be little doubt that in the main our interpretation is correct. That being conceded, to what event may we refer the chief upstrokes which in this curve often precede the chief downstrokes by a considerable interval? They are attributable to the passage of the excitation wave *past* the proximal contacts, so it seems to us. They are in reality what have been termed "extrinsic" effects, and are due to the excitation wave travelling in the neighbourhood of, but not actually under, the contacts (that part of

* It may be that the little sharp upstroke which preceded the downstroke by a very minute interval indicates this event for some cycles, though we think not (see later remarks); however, this is immaterial to the general argument.

† It cannot be beyond contact *Z* of the distal pair, for the intrinsic deflections given by this distal lead (Fig. 6. *D*) are upright, showing that of this pair the *Z* contact is always first reached.

the course which is marked by an asterisk in Fig. 1). There is a curious relation to be observed in this curve which strengthens this view: the sharpest and most prominent upstrokes in this curve tend to precede the corresponding sharp upstrokes in the lower curve (Fig. 6, *D*) by the shortest intervals (the intervals marked 0.0258, 0.0241 and 0.0227, for instance). If the excitation wave takes a circuitous course which passes the proximal contacts and subsequently reaches the distal contacts, the corresponding electrical changes at the two pairs would be separated from each other by a greater interval, the more circuitous this path. The straighter the path and the nearer its approach to the proximal contacts, the more prominent would be the upstroke in the proximal curve and the shorter would be the interval between it and the upstroke yielded by the distal pair of contacts. This relation appears to hold good, though not in its entirety.*

To sum up, the explanation which seems acceptable is that illustrated by Fig. 1. It supposes that the excitation wave takes a more or less circuitous path, passing near to, but not actually striking, the proximal contacts, and enters the area examined at some point between the two pairs of contacts: it supposes, further, that the circuitous path varies from cycle to cycle, and that both the aberration from the usual path and the cyclic variation of this aberrant path are the result of conduction changes in the auricular tissue. Whether this explanation is acceptable in its detail or not is, however, a matter of relatively small consequence. What is of consequence is that the curve may be interpreted if it is assumed that in tissue, whose conduction is already known (from observations during lower rates of stimulation, see Figs. 4 and 5 and Table, *Dog JV*) to be depressed and varying, conduction has changed progressively and now shows local irregularities, as a result of which *the path taken by the excitation wave varies*. That is the chief conclusion at which we arrive: but while we do not think that this curve is susceptible of any general explanation other than that given, we do not found our case for local irregularities of depressed conduction on this single curve, but on this and many others, some of which, taken in slightly different circumstances, will now be described.

Our conception is that curves such as that just analysed in detail are curves exhibiting *aberration* of the excitation wave in the auricle.

Further evidences of local and varying depressions of conductivity.

The experiments now to be described were performed upon the superior vena cava. A pair of stimulating electrodes is fixed to the dorsal wall of this vessel and rhythmic shocks are sent into it, and it responds regularly. A pair of leading off contacts is arranged in the line of the circumference

* The exactitude of the relation may have been upset from time to time by the form of the path taken by the excitation wave: there are a number of notches on this curve which it is not possible to explain, but which unquestionably indicate peculiarities of the cycles to which they belong.

of the vessel on its ventral surface (see Fig. 2). The excitation waves spreading from the point of stimulation in a clockwise and anticlockwise direction, meet in front. If the contacts lie a little to the left, the left contact will first pick up the excitation wave; if they lie a little to the right, the muscle under the right contact first becomes active; in the one instance the intrinsic deflections are upstrokes, in the other instances they are downstrokes. So long as the rate of stimulation is not excessive, the events are identical at each cycle; the upstrokes or downstrokes, as the case may be, occur rhythmically and are of uniform shape and amplitude. If the contacts are set as near to the mid-position as possible the curves remain quite regular and the deflections are unidirectional until the rate of stimulation is

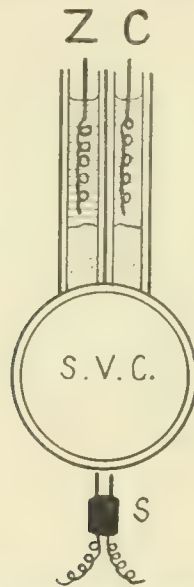


Fig. 2. A diagram illustrating experiments on the superior vena cava. A pair of stimulating electrodes (*S*) is fixed to the posterior wall of this vessel; a pair of leading-off contacts is placed upon the front of the vessel in the line of its circumference. The effects of rhythmic stimulation are recorded.

sufficiently increased; there then comes a time when conduction changes begin to manifest themselves, and very curious curves are produced. We describe three examples as illustrations.

First example. The first is shown in Fig. 8. This curve, somewhat enlarged from the original, was actually taken with an accompanying record signalling stimulation; it is not convenient to show this, as it lay on the other extreme edge of the plate; it is unnecessary to show it because the current at each stimulation affects the string, producing the sharp downward deflections which have been marked *S* in the curve. The presence of these deflections in the curve is also fortunate in that they form more secure points

for accurate measurement than do movements of the signal. Stimulation was rhythmic and at a rate of 369 per minute. But the excitation waves do not arrive rhythmically at the contacts, neither are the intrinsic deflections uniform in shape or amplitude. They change gradually or abruptly, according to the events. This strip of curve is representative of what was happening over a long period of stimulation, the events being repeated in an irregularly phasic fashion. We may first study the time interval ($S-T$) between the instant of stimulation and the arrival of the excitation wave at the Z contact; the last instant is represented by the sharp upstrokes in the curve. This interval, indicating as it does the time taken for the wave to travel from the dorsal to the ventral surface of the superior cava, changes. Near the beginning of the curve the interval is 0.0370 of a second, at the next cycle it widens to 0.0567, and during the next eleven cycles it increases further, until at last it amounts to 0.1404 of a second.* The next stimulus (marked by an asterisk) fails to propagate a wave as far as the contacts. Following the pause the interval shortens to 0.0495 of a second, and, as further stimuli succeed each other, the early events of the curve are repeated. The changes in conduction here shown are parallel with those witnessed at the $A-V$ junction, when, after progressive increase of the $A-S-U$ interval, the ventricle fails for one cycle to respond. In the present curve there is another instance of actual block: it is at the beginning of the curve (the stimulus marked by an asterisk). That the changes may be ascribed to altered conduction rate is at once suggested by this resemblance to partial heart-block between auricle and ventricle. They are not to be ascribed to change in the latency of response of the superior cava to stimulation. There are characteristics of the present curve, which show the explanation given to be correct. The deflections change their form as the intervals widen. Variable latency would not account for this: altered conduction, especially an altered path of conduction, does. There are unmistakable relations between the lengths of intervals and the forms of the corresponding intrinsic deflections, showing that the two are closely associated. If further evidence is required that conduction is varying in this curve it will be found in the form of the last intrinsic deflections of each series. The intrinsic deflection which precedes the second asterisk is a prominent upright deflection, showing that the Z contact has been reached first; the intrinsic deflection which precedes the first asterisk is small and downwardly directed, indicating that both contacts have been reached simultaneously, or more probably that the C contact has been reached somewhat earlier than its companion. This can be explained if we suppose that the rate of conduction around one side of the cava has altered more than around the other—a local variation of conduction—or that the course taken by the excitation wave has been to some extent aberrant (in this case

* An interval which, allowing 22 millimetres for the path travelled, would give a calculated conduction rate of about 150 millimetres per second. This calculation is not precise because the instants of stimulation and of the starting of the excitation wave are not exactly the same.

sinuous), an explanation which implies the same underlying local irregularity of conduction. There seems no escape from the conclusion that the excitation waves, propagated from a single point, reach the contacts by different paths. A local slowing of the conduction wave would account for such divergence or aberration; a local barrier which is not passed would similarly account for the deflection of the curve from its accustomed course. Either explanation assumes the presence of local variations in conduction.*

In the same curve there is another point of interest. Not only do the intervals *S-I* increase, but during their increase they show alternation: this alternation accounts for the coupling of intervals of almost equal length. Accompanying the alternation of intervals is an alternation in the heights of intrinsic deflections, though this is to some extent obscured by wider changes of form.

The alternation in the lengths of *S-I* intervals produces an alternation in the lengths of the inter-intrinsic intervals, which are given in seconds below the curve. The progressive lengthening of the *S-I* intervals also affects the length of inter-intrinsic intervals, as does the occasional failure of the excitation wave to reach the muscle under the contacts.

To sum up, the curve as a whole demonstrates partial block in auricular tissue responding to regular stimulation, and this partial block is of such a nature that it produces an arrhythmic activity in tissue lying at some little distance from the point stimulated. The electrogram obtained from this tissue at a distance shows irregularity, not only in the spacing of intrinsic deflections, but also in their form: this last change is to be explained only if we assume local variations in the degree of block, whereby the wave proceeds by varying paths to the contacts. All these disturbances are attributable to one underlying factor, namely, changes in conduction, induced by the rapidity of stimulation. It is to be remarked that cessation of stimulation at once and repeatedly brought the rapid action of the auricle to an end; thus it is shown that the disorder was confined to excitation waves propagated directly from the artificial stimuli entering the auricles.

Second example. The second example was obtained under similar experimental conditions to the first. Two strips of somewhat enlarged curves are shown (Figs. 9 and 10). Stimulation was at the rate of approximately 300 per minute, and in the opening period of the first curve (Fig. 9) the events under the contacts do not vary. The *Z* contact is first reached; witness the steep upright deflection. Each stimulus is seen in the curve; it is marked by a diminutive spike (*S*); this is shortly followed by an extrinsic deflection (*E*), a dip from which the main or intrinsic deflection (*I*) rises. Two series of readings are given above this curve; those written horizontally are the inter-intrinsic intervals, those written vertically are the intervals

* The curve now described is one of several from the same animal, in one of which short periods of 2:1 block were exhibited.

between the stimuli and the corresponding intrinsic deflections. Below the curve are written vertically the intervals between the stimuli and the corresponding extrinsic deflections. At first the events are orderly: each stimulus is followed at about 0.018 of a second by the extrinsic deflection, and at about 0.030 of a second by the intrinsic deflection. Throughout the whole curve each stimulus is followed by an extrinsic deflection at a constant interval, but towards the end of the curve the intrinsic deflections begin to vary in form, and the last two cycles show an obvious reversal of direction (cycles *k* and *l*). In other words, while the excitation waves of the early cycles reached the *Z* contact first, those of the last cycles reached the *C* contact first. The explanation is not in doubt. Each stimulus excited a wave from the dorsal surface of the cava, and this was propagated after a constant interval and in a constant direction: this is shown by the constant extrinsic deflections* which succeed each stimulus. Nevertheless, the excitation wave ultimately reaching the contacts reaches them now from one side, and now from the other side of the cava, and, as this change reaches its height, the extrinsic and intrinsic deflections alter their time relations to each other.

The second curve (Fig. 10) is from the same experiment, and was taken under identical conditions: it shows similar events, but is even more demonstrative. Not only do the extrinsic deflections become detached from the intrinsic deflections, a process which begins at cycle *j*, but one, namely, the extrinsic deflection of cycle *l* stands completely isolated. Here is conclusive evidence that the usual path taken by the excitation wave is barred at some point between the dorsal and ventral surface of the cava. Considering both curves together, the detachment of the extrinsic from the intrinsic deflection or the isolation of the former is attributable to a local block. The extrinsic deflection represents the activity of muscle lying at some little distance from the contacts, the intrinsic deflections represent activity of muscle beneath the contacts. The excitation wave starts from the point of stimulation in a constant fashion at each stimulus, but it does not proceed in a regular fashion beyond a certain point: it reaches a line which it is unable to cross, a line on the side of the cava towards which the *Z* contact is placed: and the excitation wave, to reach the contacts, pursues the other channel open to it, namely, the side of the cava towards which the *C* contact lies. For this reason the arrival of the excitation at the contacts is delayed (witness the increase in the inter-intrinsic intervals at the change). The delay is, however, sometimes too great to be entirely accounted for in this fashion. Naturally, when the decrease in conduction comes it affects both sides of the cava, although the temporary block may be at times absolute on one side only, at times absolute on both sides.

* Extrinsic deflections failed to show in the curve previously described; had they shown they would have formed an immediate and overwhelming evidence against altered latency of response to stimulation, as they do in the present curve. The extrinsic deflections come from active muscle, and this activity is the result of the stimulus in each cycle: the first muscle excited is excited at a constant interval after the stimulus enters.

Third example. The third example is of a simpler kind. It was obtained by a similar method, though with this difference; the contacts lay, not in the circumferential line of the cava, but in the length of the vessel. Stimulation of the dorsal surface of the cava at the rate of approximately 460 per minute produced a curious form of alternation (Fig. 7). The excitation waves reach the contact almost rhythmically. The chief feature of the curve is alternation in the direction of the intrinsic deflections. They are steep deflections and of considerable amplitude, but they are directed alternately upwards and downwards. This is due to the excitation wave alternately reaching the Z and C contacts first; a disorder which is to be explained only by assuming that the path to the contacts varies in alternate cycles as a result of local changes in conduction. The example is of special interest because it demonstrates that, although the path taken by the excitation wave, in travelling from the point of stimulation to the point investigated, may vary from beat to beat, yet the excitation waves may arrive at this last point at almost regular intervals.

The end of stimulation is shown in this curve; it is followed by an after-effect, the two beats marked by asterisks. The presence of this after-effect does not affect the interpretation of the rest of the curve, for each excitation of the tissue under the contacts clearly follows upon one of the rhythmic stimuli (see signal, below the curve).

2. -- After-effects.

Before describing those examples of after-effect in which the beginning or ending of this effect has been recorded, certain relations between the nature of after-effects and the conduction changes already described are to be noticed, for these relations seem to be of not inconsiderable consequence theoretically. After-effects following rhythmic stimulation of the auricle are of three kinds: (1) The most frequent is fibrillation of the auricles.* (2) The occasional after-effect is pure flutter, the condition dealt with in the preceding article. (3) Another form of after-effect, which is frequent, is what may be termed "impure flutter," a mechanism having the same basis as pure flutter, but disturbed in a peculiar manner. This mechanism will be dealt with fully in a succeeding article; suffice it for the moment to say that it seems to be transitional between pure flutter and fibrillation, and that it is the most unstable of the three types.

Now, it is the rule to find a constant, or almost constant, form of after-effect in a given animal. In one animal the after-effect is flutter, in another it is fibrillation; we may speak, therefore, of this auricle as an auricle prone to fibrillation, and of that auricle as tending to flutter. There are exceptions, it is true; thus, an auricle which has been for many hours under observation may change its habit; in other instances, and these are

* The term fibrillation is used in this article to designate conditions which give rise to constantly irregular oscillations in lead II.

not uncommon, an auricle which exhibits flutter or fibrillation as a usual after-effect, may from time to time exhibit impure flutter. These exceptions are not of such a kind, nor so frequent, as to invalidate our dividing auricles into the two broad classes, namely, those disposed to flutter and those prone to fibrillate. These tendencies have been indicated in the head lines of the foregoing tables, and are exemplified in the notes which follow each table.

The subdivision is important, for the following further generalisation seems justified, in the main, at all events. There are auricles which show, when submitted to rhythmic stimulation of increasing rate, only a brief period in which the conduction rate can be ascertained to decrease: these are the auricles in which, once the conduction rate begins to fall, very slightly higher rates of stimulation yield relatively complex disturbances, interpreted as due to aberration in the auricle. The small range of rate, over which increased transmission times can be measured, is in fact attributable to the onset of complex changes of conduction at higher rates. Auricles which behave in this fashion are auricles prone to show fibrillation as an after-effect of stimulation.

On the other hand, there are auricles in which the rate of stimulation may be increased over a wider range after a decrease in the conduction rate has appeared, and in these the transmission intervals widen out gradually and progressively without material variation from cycle to cycle as the rate is increased. Auricles which behave in this fashion are auricles prone to show flutter as an after-effect of stimulation.

To put the whole relation of after-effects more pointedly, it may be said that when the auricle is submitted to rhythmic stimulation at increasing rates, there comes a time when conduction begins to fail. *If this failure of conduction continues to be relatively uniform in its degree in the tissue examined and constant from cycle to cycle, then, as the rate is further raised, the end result of stimulation will be flutter. On the other hand, if the failure soon displays itself in a manner interpreted as due to regional inequalities of depressed conduction with variations from cycle to cycle, the end result of stimulation will be fibrillation.* The first observation is consistent with our new knowledge of flutter, for, as is shown in the last article, it is a condition in which conduction rates are lowered, but in which there is no variation of conduction from cycle to cycle. The second observation points clearly to the nature of fibrillation, a subject which will be pursued at a later date.

After-effects of stimulation are always witnessed, providing that the rate of stimulation is driven high enough and stimulation is repeated. They are especially associated with the changes in conduction which we have described: for, whenever the range of rate, over which conduction changes may be demonstrated to occur, is entered, then short or long after-effects are seen.

Very brief after-effects. We may now pass on to describe certain characters of after-effects and to examine briefly their relation to the theory

of circus movement. The auricle is stimulated by means of rhythmic shocks at advancing rates; stimulation at a given rate is maintained for from 10 seconds to half a minute; if, after several repetitions, the auricle shows no tendency to continue its rapid action when stimulation ceases, the rate of stimulation is raised and the observations are repeated. Sooner or later short after-effects are seen. The after-effect may comprise one, two, several or many cycles; as with longer after-effects (either flutter or fibrillation), their appearance when stimulation is withdrawn is uncertain. The auricle may be stimulated several or many times at a particular spot without an after-effect being witnessed; a final stimulation is followed by an after-effect of shorter or longer duration. The length of the after-effect is uncertain; much more often than not it consists of a few beats, occasionally it lasts for a few or many minutes. The short after-effects, like the long, tend to be of one type in a given animal, and this type seems to be independent of the rate or strength of the stimuli and to be independent to a lesser extent of the point stimulated. A résumé of some short after-effects is given in the accompanying table. The chief features may be illustrated by a description of characteristic curves. Over the first half of Fig. 11 the auricle is responding to rhythmic stimulation at a rate of 432 per minute. The stimuli were applied at the cephalic end of the tania terminalis, and curves were taken from lead *II* and from a direct lead simultaneously. The contact of the direct lead lay on and in the line of the tania terminalis in the mid-caval region, the *Z* contact being nearest to the point stimulated. The auricle responds to each stimulus, and uniformly, with the exception of slight alternation in the heights of the deflections and of the inter-intrinsic intervals.* This alternation bears witness, as we have seen, to conduction changes in the auricular tissue. The auricular cycles have an average length of 0.1377 of a second up to the point where stimulation ceases. Following the latter event, there is a pause measuring 0.1895 of a second; the auricle then beats spontaneously. This single beat (marked by an asterisk) alone constitutes the after-effect; the beats which follow belong to the restored heart rhythm.

It is to be noticed that the cycle terminated by the after-beat is very distinctly longer than the preceding cycles (responses to stimulation).

Fig. 12 is from the same animal and was taken under the same conditions. It is very similar to the last, but the after-effect here consists of two cycles.† The first has a length of 0.1927 and the second of 0.1851 of a second. Fig. 13, which is also from the same animal, exhibits an after-effect of four cycles; their lengths are 0.2153, 0.1605, 0.1548 and 0.1626 of a

* The stimuli were regular; there is a corresponding alternation in the stimulus to intrinsic intervals. These measurements have been made from the curve of the direct lead in which each induction shock shows as a minute upward deflection. These points are more reliable than the signal below the curve.

† Similar after-effects were witnessed in an experiment of Levy's, in which a cat's ventricle was submitted to rapid rhythmic shocks. He attributes them to a circulating mechanism.¹

Table illustrating short after effects of rhythmic stimulation.

Cycles in response in seconds.					Cycles of after effects, in seconds.				Cycles of restored rhythm, in seconds.				Record No.
1	2	3	4	5	1	2	3	4	1	2	3	4	
.2252	.2297	.2265	.2273						.4076	.3931	.3963	.4787	KM 2
.2055	.2058	.2004	.2042						.3504	.6803			KM 10
.4418	.4422	.4410	.4430						.3336	.4751	.4483	.4451	KL 2
.4833	.4820	.4812	.4805	.4794					.3291	.4706			KD 28
.4633	.4622	.4633	.4616		.2039				.9377				KD 113
.4277	.4362	.4294	.4297		.1987				(.3500) (.1123)	.4049			KD 19*
.4347	.4400	.4375	.4388	.4378	.1895				.660	.4304	.4026		KD 21*
.4380	.4400	.4383	.4411	.4348	.1927	.1854			.3520	.4498	.4258	.4225	KD 23*
.4980	.4938	.4926	.4942	.4904	.2153	.4605	.4548	.4696	.4948	.3223	.4635		KD 24*
.4692	.4471	.4666	.4486	.4554	.1734	.1622			.4729	.4689	.4652	.4627	KL 1*
.4319	.4344	.4305	.4357	.4298	.1734	.1669	.1367	.2604	.5740		.4652	.4624	KD 24
.2927	.3005	.2962	.2995		.2330				.4055	.4762	.4624		KL 4*
.4439	.4469	.4465	.4443		.4574				.3840	.3433	.6044	.3620	KC 24
									.4420	.3594	.3889	.3872	KC 24
.4554	.4554	.4564	.4584	.4780	.1740				.4457	.4426	.4669		KC 31*
.5079	.5056	.5100	.5055		.5144				.4295	.3438	.2967	.2732	KC 44

* The animals from which these curves were obtained yielded long continued flutter on repeated stimulation of the auricle at increasing rates.

† This animal yielded fibrillation as a longer after effect, but was under esame.

‡ This animal also yielded short periods of irregular flutter on after effects.

second. In all these examples,* the length of the first cycle of the after-effect is very similar (namely, 0.1895, 0.1927 and 0.2153 of a second). Fig. 14 is from another animal and the events are parallel. During the response to stimulation the auricular cycles measure in the average 0.1325 of a second, the first cycle of after-effect measures 0.1731 of a second, the next cycle measures 0.1622 of a second. Other examples will be found in the table. Thus, it is a rule that the first cycle of the after-effect is longer than the cycles during the period of stimulation, and this is consistent with the rule that the rate of flutter, when this forms a longer after-effect, is lower than the rate of stimulation which induces it.† It is not difficult to explain this lengthening if we assume, as we must, that in these short after-effects we are dealing with essentially the same phenomenon as in pure flutter, namely, circus movement. During the period of stimulation the length of the cycles is governed by the rate of stimulation: during the after-effect it is governed by the rate at which the circus movements are completed. It happens in most experiments that the rate of stimulation inducing those changes in the auricle upon which the initiation of flutter depends must be at least as great as the rate of the flutter itself: a lower rate of stimulation does not suffice. Consequently, the rate of stimulation actually used is almost always in excess of the rate of the succeeding after-effects. This excess of rate does not militate against the appearance of flutter as an after-effect: on the contrary, it seems to favour it, for reasons which will be alluded to in a later article. Very occasionally it will happen that the rate of stimulation used corresponds to the rate of the after-effect: two examples, in which the length of the after-effect cycle is equal to the cycles of stimulation, are shown at the end of the table (*KU*, record 31, and *KC*, record 1).

To come to the next point, it has been seen that the second cycle of the after-effect is shorter than the first: that has always been so in our observations. This also may be explained without difficulty if we assume that we are dealing with a circus movement: for the rate of the auricle in this circumstance depends on the time taken for the circuit to complete itself, and the latter will depend upon the state of conduction in the tissue. We are dealing with rates of beating which are already influencing this conduction: a rise of rate lowers and a fall of rate augments the conduction rate. During the period of stimulation, conduction is more strained; consequently the first circus movement of the after-effect will take place comparatively slowly: this first slow circuit itself induces some recovery of conduction, and consequently the succeeding circuits are accomplished more quickly and the corresponding cycles are shorter.

* These three records were taken from *Dog KD*, which yielded to similar stimulation long periods of flutter, described in detail in Part II of the present series of articles.

† Of this many instances will be found in our description of experiments, in this and the preceding article. It is a rule to which there are occasional exceptions, which will be noticed at a later stage.

If the curves already described are examined, a curious phenomenon is to be seen when the after-effect terminates. It is followed by a cycle which is of greater length than the cycles of the after-effect, but which is of shorter length than the cycle of the restored rhythm. At first the cycle in question might seem to belong to the after-effect itself, but we believe that this is not actually the case. Thus, in Figs. 12 and 13, in which the phenomenon is well shown, the deflections which correspond to the beat terminating these cycles (marked in the direct leads by asterisks) depart from those of the after-effects proper in their form. Generally speaking, they resemble in form the deflections of the restored beats very closely: the same resemblance is witnessed in the auricular complexes in lead *II*, and in this it is more convincing: but as the auricular complexes often fall with preceding ventricular complexes, as in the case in Figs. 12 and 13, they are not always clear in lead *II*. Not infrequently, the outline of the complexes in lead *II* is clearly defined, and then it becomes quite evident that the cycle in question is terminated by a heart beat originating from the pacemaker. Thus in Fig. 15, which is an example of the kind, there is no true after-effect. Stimulation ceases and the normal heart rhythm is at once restored. The first auricular complex of this restored rhythm (*P*) is perfectly natural in outline, resembling closely the succeeding auricular complexes: the same resemblance is seen in the deflections of the direct lead. In this case there can be no doubt that the beat marked by an asterisk belongs to the normal rhythm of the heart: but the lengths of the cycles should be noted. The cycle which precedes the marked beat has a length of 0.4076 of a second, the next one has a length of 0.5931 of a second. From this point onwards the cycles shorten. Other examples are entered in the table (*KM*, 2, to *KD*, 28), in which a similar increase is found in the length of the second beat of the restored rhythm as compared with the first, and the same quickening of the rate follows this disturbance.

If the beats, which are marked by an asterisk in Figs. 12-14, were regarded as belonging to the after-effect, we might find it difficult to explain them by the theory of circus movement: but that they do not so belong is in many instances clear, and in the remaining instances is strongly suspected. The difficulty of explaining them as parts of the after-effect is our chief reason for discussing their origin at some length. Sometimes, as in Fig. 11, these short cycles are not seen. The restored auricular rhythm shows curious irregularities in its first phases, which it is not our present purpose to explain.*

Onset of flutter. Of records showing the onset of long continued flutter we have but few examples. They are difficult to obtain. An illustration is given in Figs. 16 and 17. The after-effect of these two records (the first

* The interpretation of the ends of these after-effects is also complicated by the irregularity of the site from which the restored beats spring. They do not always come from the sino-auricular node.

taken to cover, and the second taken $1\frac{1}{2}$ minutes after, the end of stimulation) lasted 3 minutes. The superior vena cava was stimulated with rhythmic shocks at a rate of 484 per minute: the direct lead, shown above, was from the upper reaches of the tania. The intervals from the signal marks of stimulation to the intrinsic deflections* are sufficiently constant to render it certain that the auricle is still responding for the most part to these stimuli. When stimulation ends, a longer cycle (0.1548 of a second) appears. The next cycle is shorter (0.1385 of a second). This change is precisely comparable to those which were described for the brief after-effects of Figs. 12, 13, etc., and is subject to the same explanation. The flutter now continues, quickening a little in rate and being in a very slight degree irregular.† When the next record (Fig. 17) was taken ($1\frac{1}{2}$ minutes later) this slight irregularity had disappeared, the rate having become a little slower and steady.

After-effects following single impulses. Fig. 18 is an exceptional, but interesting record, taken from the same animal as Figs. 11-13, though at an earlier period of the experiment. The direct lead was from the mid-caval region and the right auricular appendix was stimulated rhythmically. The rate of stimulation was slower than that of the previous figures. The last four responses to stimulation are seen at the beginning of the record. After the last stimulus has entered the muscle, a single beat (*a*) appears as an after-effect.‡ The normal heart rhythm is then resumed, but this is interrupted by premature beats (*b*, *c* and *d*). The interesting feature of the curve is that these premature beats follow the normal beats, with which they are coupled, by intervals which are practically identical with the cycle preceding the after-beat (*a*). The intervals are 0.1922, 0.1926, 0.2002 and 0.1960 of a second. It is true that the deflections *a*, *b*, *c* and *d* are not identical in form, but the uniformity in the lengths of the cycles which they terminate strongly suggests that they have all arisen in similar fashion. Now the beat *a* is an after-effect in every way comparable to those of Figs. 11-13, and these must be regarded as comparable to the first beats of a continuous flutter. It is difficult to avoid the conclusion that the premature beats of this curve (*b*, *c* and *d*) are also attributable to the same cause, namely, circus movement. But if that is the case we must acknowledge that a circus movement may be set up by a natural heart beat. It is to be impressed, however, that, while this curve represents one of a number of very similar ones obtained from this animal (Fig. 19 is a second example),§ the premature beats were confined to periods immediately succeeding rapid stimulation: therefore, they were in some way connected with it. It seems possible that conduction, a depression of which is a chief underlying factor in establishing

* These are written vertically below lead *II* in the figure.

† Slight unsteadiness in the very early stages of flutter is not uncommon.

‡ This cycle has a similar length to those seen in Figs. 11-13, but, since the rate of stimulation is lower, it is of much the same length as the cycles of response.

§ There is in this curve no after-beat, and there is a greater divergence in the lengths of the cycles which the premature beats (marked by asterisks) terminate.

flutter (circus movement), is in certain circumstances depressed with unusual ease directly after a period of rapid heart action, and that in such circumstances a single beat of the auricle may be enough to establish those conditions under which the first beat circulates and re-enters muscle through which the first has passed.*

That flutter itself may appear without the intervention of rapid preliminary impulses seems clear from observation. We have seen it established in very susceptible animals on striking the auricle a single and slight blow with the handle of a knife: we have seen it follow a single induction shock: but these examples are very rare. An auricle† which showed this curious susceptibility was stimulated by means of rhythmic shocks at a rate of 469 per minute, applied to the mid-caval region. Pure flutter followed, and this lasted for many minutes. Every now and then, however, a slow action of the heart was resumed for one, two or three cycles: then flutter started again spontaneously. On re-stimulating the auricle, it broke back at once to a slow rhythm, and after this had proceeded for a few cycles flutter appeared spontaneously. A record of this event was obtained, and is shown in Fig. 20. When stimulation ceases in this curve no after-effect follows, but the auricle beats thrice in response to impulses originating from an abnormal centre (the three corresponding complexes are marked *P* below the curve). Following immediately upon the last of these beats, the auricle passes into flutter‡ spontaneously.

To sum up, the usual manner in which flutter begins, when examined in detail, is not only compatible with the theory of circus movement, but lends a good deal of support to the theory. Spontaneous flutter, following a single stimulus, or breaking in upon a slow heart rhythm, we are not at present prepared to explain: the phenomenon is parallel to the occasional appearance of flutter following upon rhythmic stimulation at rates lower than that of the subsequent flutter.

The ending of flutter. Records of the end of flutter of any considerable duration are not easy to obtain, and we have not many examples in which the events are clearly displayed. In one animal rhythmic stimulation produced periods of flutter lasting in the average several minutes. The end of one of these after-effects is shown in Fig. 21. The direct lead was taken from the mid-caval region, the *Z* contact lying toward the inferior cava. The general movement of the excitation waves was down the tænia while the auricle fluttered; the intrinsic deflections are directed downwards, indicating that the *C* contact first received the wave. Apart from a slight

* The example being exceptional requires a special explanation, and is in itself insufficient, of course, to be more than suggestive.

† A long period of pure flutter, obtained from this animal (*K2*), was described in our last article. The flutter was similar to that now described, in that the auricular complexes of the two periods were similar; in both periods also the excitation wave passed upwards through the tænia terminalis.

‡ The flutter is impure in this the earliest stage of its progress.

alternation in the lengths of the inter-intrinsic intervals, the mechanism is a perfectly uniform one. The flutter ends without preliminary disturbance. It is followed by a cycle having a length of 0.3326 of a second, the next cycle is longer, the third shows shortening; these changes in the restored rhythm are very similar to those displayed by Fig. 15 and by other figures already described. The last auricular complex of the period of flutter does not appear to be peculiar: thus, the two auricular complexes marked by asterisks, which may be compared because they fall at similar times in the ventricular systole, are of the same form. The deflections in the direct lead are of constant form and amplitude throughout. The ending is abrupt, and there is in the curve no visible cause of it. By the theory of circus movement, the ending could be accounted for if we assumed the development of a local barrier (block) on the path of the central wave, which brought the latter to a standstill. If we accept the conclusion that flutter is due to a circus movement there seems to be no way in which it can terminate but by a change of conduction in some part of the central path. Although Fig. 21 shows no sign of such change, other examples appear to do so, and of these Fig. 22 is the most notable example which we possess. Flutter was induced by stimulating the inferior cava with rhythmic induction shocks at a rate of 518 per minute, and it lasted 1 minute and 10 seconds.* The last seven regular cycles of this flutter are shown at the beginning of the figure. It is to be noticed that they shorten a little as they proceed; the increase of rate is perhaps responsible for what follows. In the curve of the direct lead a cycle of 0.2223 of a second appears, and this is followed by two shorter cycles of unequal length. During the earlier phases of regular action the intrinsic deflections (*I*, *2*, and *3*) stand in constant relation to the auricular complexes of lead *II*: so they do in the later stages (deflections *6*, *7* and *8*). But the actual time relation has altered; the interval has shortened from 0.06 to 0.025 of a second. The intrinsic deflections of the last phase have come to occupy a later position relative to the corresponding auricular complexes. That fact in itself points clearly to a lowering of conduction in this last phase. Such a disturbance might be limited to the centrifugal path; actually it can be shown that the central path has been affected. *P1* to *P4* form a regular series of complexes, but *P5* is delayed.† It should begin where the dotted line (marked by an asterisk) has been drawn across the curve. The succeeding auricular cycles in lead *II* are also of unequal length (*i.e.*, the intervals between *P6* and *P7* and between *P7* and *P8*). Thus, in its terminal phases, the curve shows clear evidences of disturbance involving the central path;

* A long period of flutter obtained from this animal (*KQ*) has been described in our previous article. In this long flutter the auricular impulses were similar to those seen in the present record, and the excitation waves were passing up the *tēnia* as on the present occasion. The direct lead in Fig. 22 was from the upper reaches of the *tēnia*, the *Z* contact lying towards the inferior vena cava.

† This complex (*P5*) also alters its form; for that reason it is difficult to measure accurately, and the figures in seconds are omitted; but there is no doubt as to its delay, which is shown by coarse measurement.

such disturbances can hardly be explained except on the ground of altered conduction. To this altered conduction we attribute, in the present instance, the termination of the flutter. Disturbed or impure flutter, a condition which will be examined more fully in the succeeding article, is unstable.

In Fig. 23 the auricle is responding at first regularly* to rhythmic stimuli up to the moment when stimulation ends. The after-effect begins (as in Fig. 16, etc.) with a longer cycle (0.1731 of a second) and there then follow cycles of irregular length. The second of these is terminated by two deflections (marked by an asterisk), the one directed downwards and the other upwards. This double effect is of a type which we have already recognised (Fig. 6), and have interpreted as being due to a local variation in conduction.

To sum up, flutter ends abruptly, and those records which seem to display the cause of the termination appear to us to display evidences of local variations in the conduction of the excitation wave. The manner of its termination is compatible with the theory that flutter is constituted by a circus movement in the auricle.

SUMMARY AND CONCLUSIONS.

1. When the mammalian auricle is stimulated by means of rhythmic induction shocks, and the rate of stimulation is gradually raised, the speed at which the excitation wave traverses the muscle shows no appreciable change over a considerable range of rate.

2. When the rate of response rises to about 300 or 450 per minute the transmission intervals widen. In some auricles this widening progresses uniformly as the rate of stimulation rises higher, while the muscle still responds regularly: these auricles are prone to flutter as an after-effect of stimulation. In other auricles, the fall of conduction rate is soon followed by irregular conduction: these auricles tend to fibrillate as an after-effect of stimulation.

3. Electrograms are described, which are interpreted as illustrating irregular conduction and the development of local barriers, of a temporary nature, in auricular tissue responding to high rates of stimulation.

4. After-effects of rhythmic stimulation of the auricle are described. These may be very short or longer. The last consists of pure flutter, impure flutter or fibrillation. The rate of beating during an after-effect, consisting of flutter, is generally lower than the rate of stimulation which provokes the after-effect. The brief after-effects and flutter itself both end abruptly;

* Slight alternation in the lengths of the cycles is present.

the latter may show evidences of locally disturbed conduction at its termination. The events at the beginning and ending of flutter are consistent with, and therefore support, the theory that flutter is due to a circus movement in the auricle.

5. Sometimes flutter may be produced by stimulating rhythmically at a slower rate than that exhibited by the flutter itself. Flutter in certain circumstances may follow immediately upon spontaneous slow heart beats.

6. A rare effect of rhythmic stimulation is the subsequent appearance of coupled beats: the second beat of the couple shows affinities to beats constituting after-effects of stimulation.

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LEWIS and MATHISON. *Heart*, 1910-11, II, 47.
LEWIS, MARSH and WHITE. *Phil. Trans. Roy. Soc.*, 1914, B, CCV, 375-420.

Explanation of the Figures.

Some of the following figures have been a little reduced in scale. In the originals the horizontal lines are a millimetre apart.

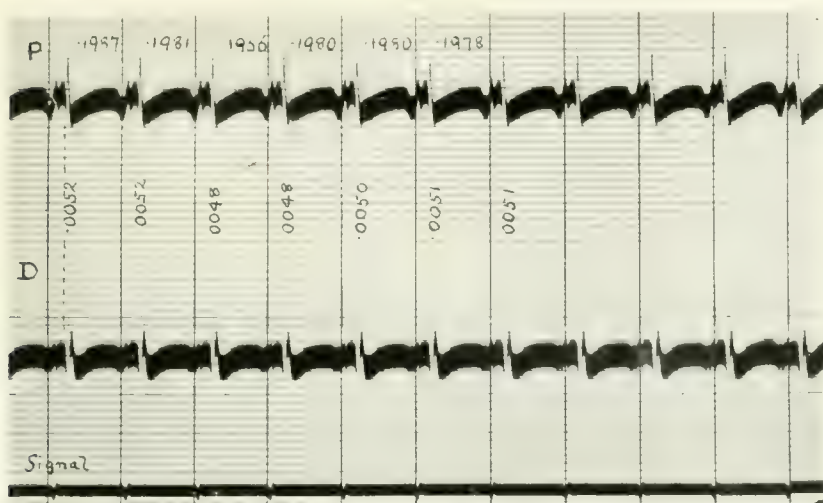


Fig. 3. *Dog K V.* (Record 18.) Simultaneous electrograms, from two pairs of contacts lying in one line with a point of stimulation on the body of the right auricle (see Fig. 1). *P* = curve from proximal and *D* = curve from distal pair of contacts. The inter-intrinsic intervals are written horizontally in decimal points of a second; the intervals between proximal and distal intrinsics are written vertically. The signal of stimulation is shown at the bottom. The heart is responding regularly at a rate of 303 per minute and the conduction rate from beat to beat is unvarying. The standardisation of these curves and those which follow was approximately 5 millimetres = 3 millivolts. The time lines represent fifths of a second.

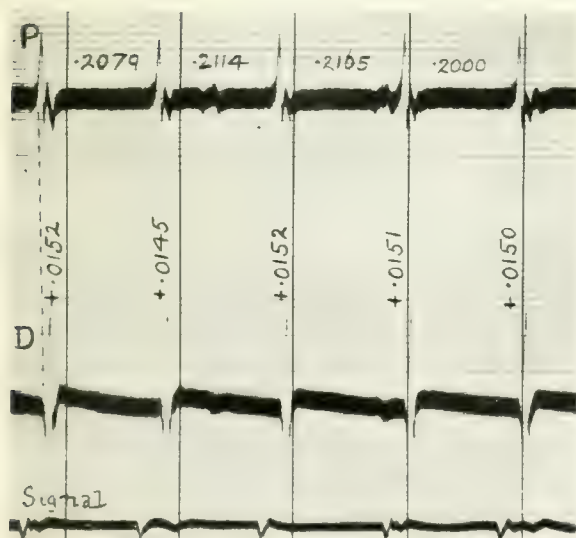


Fig. 4. *Dog J V.* (Record 6.) Similar curves from another animal; the rate of stimulation is 287 per minute. The heart responds and the transmission interval is 0.0150 of a second. When this curve was taken the shocks were not entering the auricles quite rhythmically.

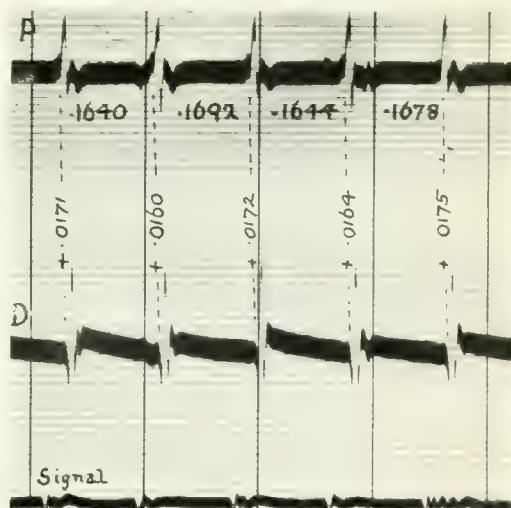


Fig. 5. *Dog J V.* (Record 8.) Similar curves from the same animal, the rate of stimulation having been raised to 361 per minute. The heart responds, but the conduction rate has now fallen a little. It also alternates slightly from cycle to cycle. The inter-intrinsic intervals show a sympathetic alternation in length.

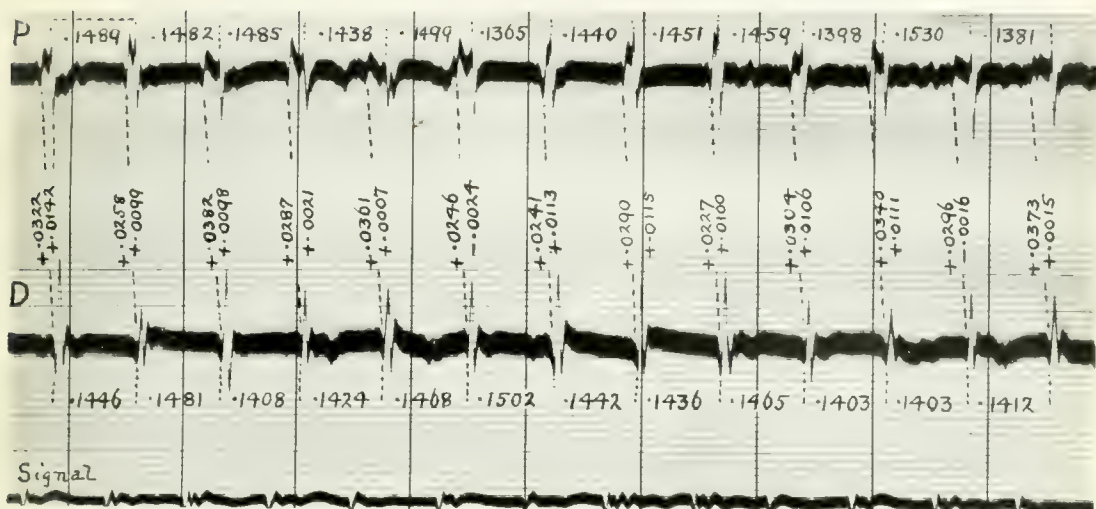


Fig. 6. Dog J.V. (Record 11.) Similar curves from the same animal, the rate of stimulation having been raised to 417 per minute. A degree of inco-ordination has developed. The inter-intrinsic intervals of the distal curve (D) vary a little, and the deflections vary in height. The proximal curve shows profounder changes, which are fully described in the text. The curve illustrates what is believed to result when conduction varies locally.

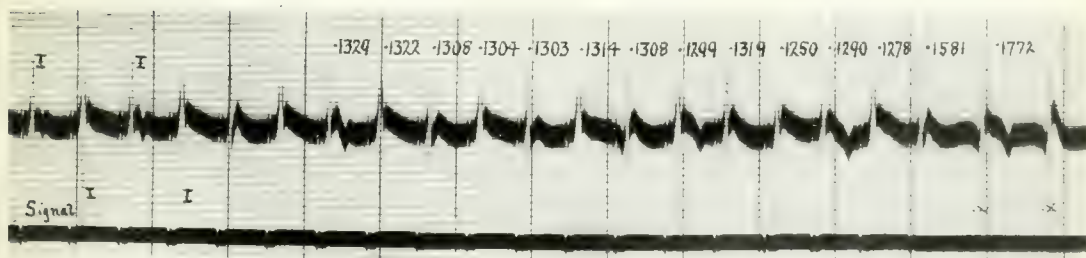


Fig. 7. Dog KW. (Record 17.) An electrogram from the ventral surface of the superior cava, responding to dorsal stimulation. In this instance the contacts lay in the line of the cava. At alternate beats the excitation wave reaches Z and C contacts, but the inter-intrinsic intervals vary only slightly. Two cycles of an after-effect are shown (cycles marked by asterisks). The signal of stimulation is shown below.

Fig.

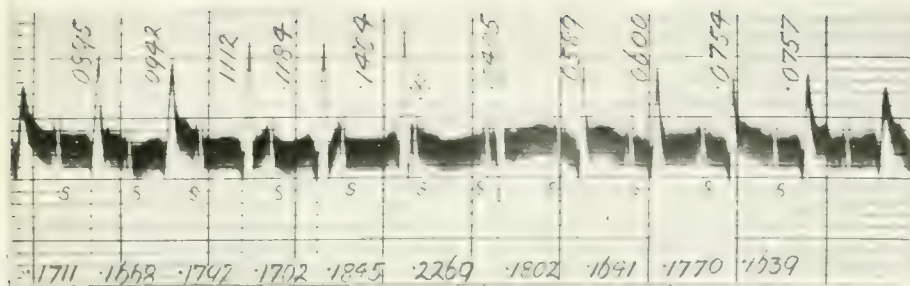


Fig.

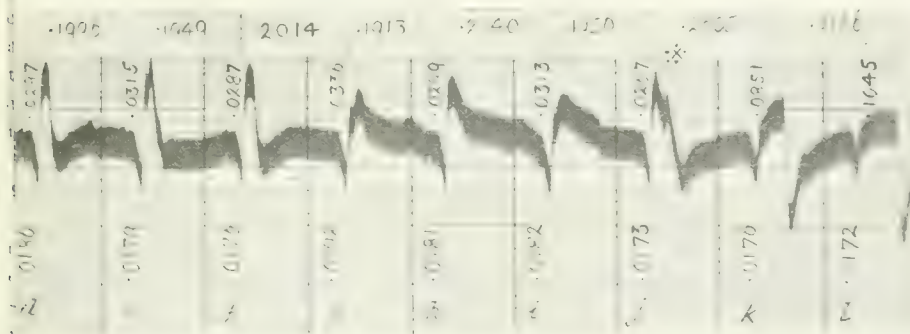
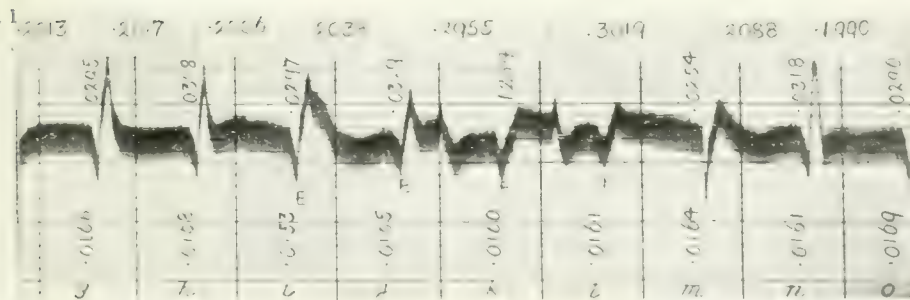


Fig. 1



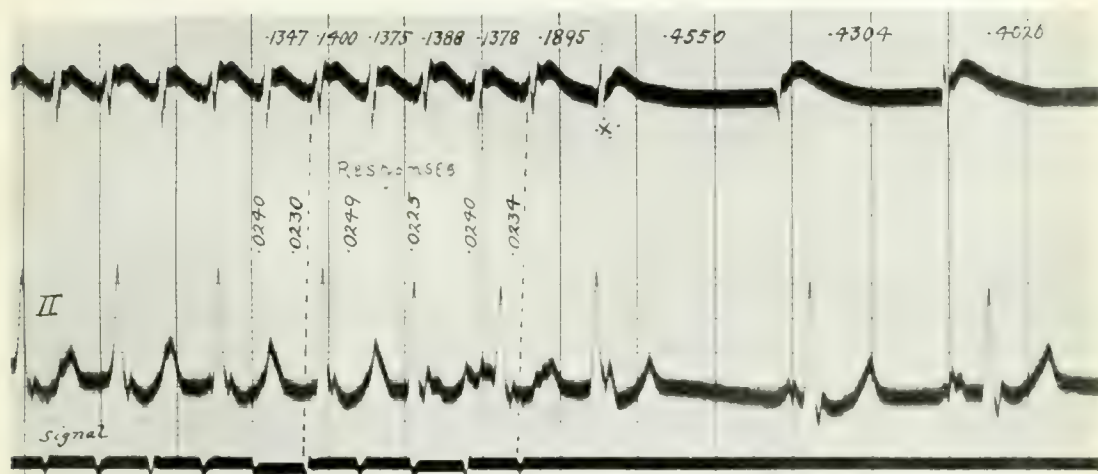


Fig. 11. *Dog KD.* (Record 21.) Electrogram from mid-caval region (Z contact above) and electrocardiogram (lead II). Showing the end of response to rhythmic stimulation at the upper end of tena terminals and a single beat as an after-effect (see signal). The figures written vertically are the intervals between stimulus (measured in the electrogram) and corresponding intrinsic deflections. For this and the succeeding curves the standardisation of the electrogram was 3 to 5 millivolts = 3 millimetres, and of the electrocardiogram (lead II) it was 1 millivolt = 1 centimetre.

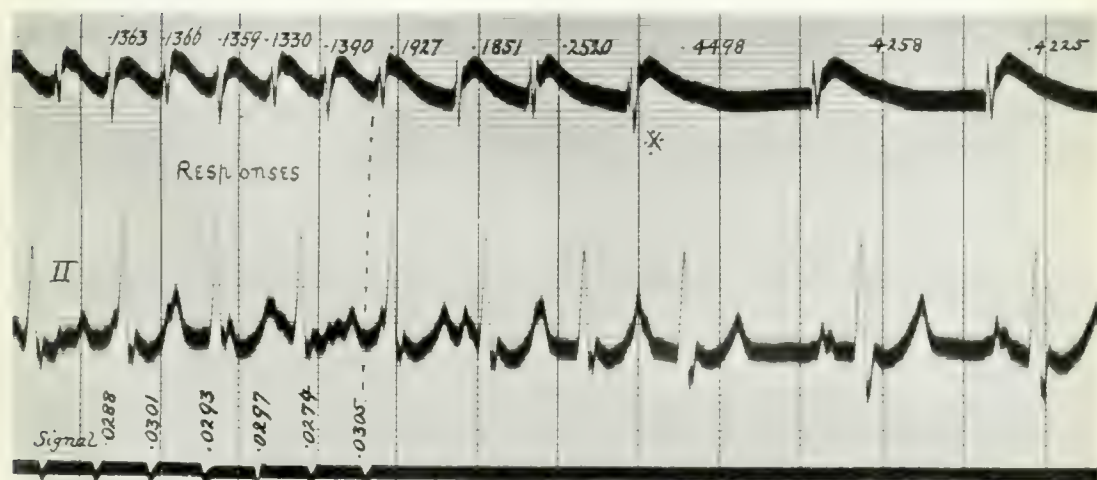


Fig. 12. *Dog KD.* (Record 23.) Similar curves from the same leads, showing an after-effect of two beats. The intervals, between stimuli and intrinsic deflections, were measured in the electrogram.

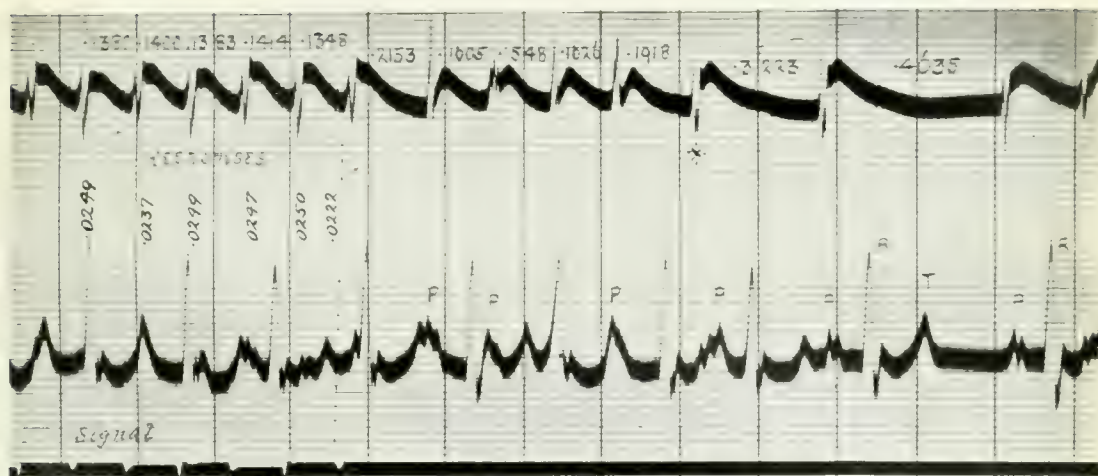


Fig. 13. Dog KD. Record 24. Similar curves and measurements. Showing an after-effect of four beats.

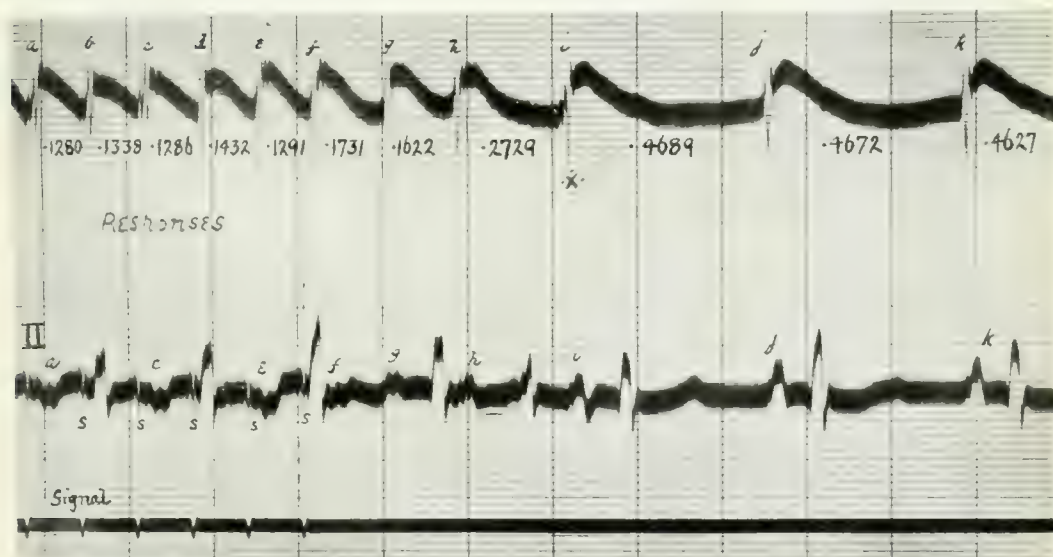


Fig. 14. Dog KE. Record 11. Similar curves from another animal, showing an after effect of two beats. The electrogram was taken from a mid-caval lead (Z contact above); stimulation of inferior vena cava. S = deflections due to current of stimulation.

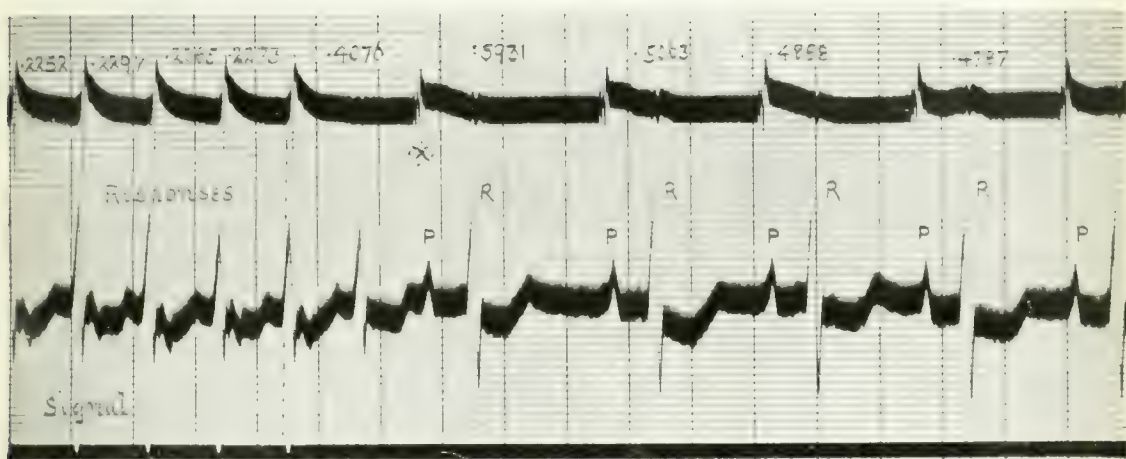


Fig. 15. *Dog K M. Record 2.* An electrogram from the mid caval lead, Z contact (above) and an electrocardiogram from lead I L. The record shows the end of rhythmic stimulation at the inferior vena cava and the return of the natural heart rhythm. Note the relatively short pause which precedes the first beat of the normal rhythm.

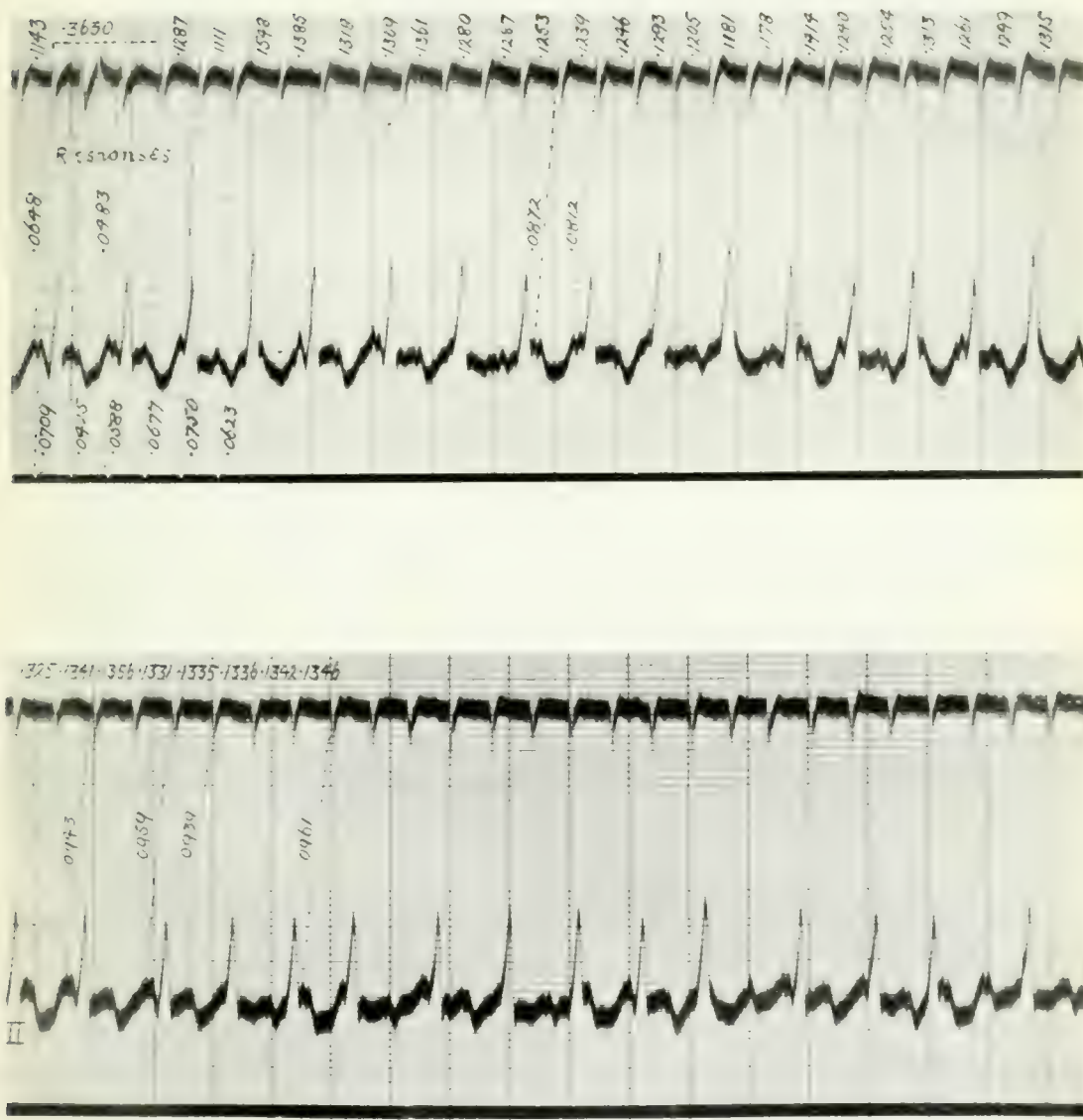


FIG. 16. 117. D. K. Q. R. 135011 and 122. Electrogram from the base of the right atrium, near the tricuspid contact below, and curve from lead II, showing an after-effect of stimulating the inferior vena cava. Fig. 16 was taken to show the end of stimulation, and Fig. 17 was taken 1½ minute later. The after-effect lasted 3 minutes. The inter-intrinsic intervals are written above the electrogram. The figures written vertically between the two curves are intervals between auricular complexes in lead II and intrinsic deflections in the electrogram. The figures written below the electrocardiogram (Fig. 16) are the intervals between signal marks and intrinsic deflections.

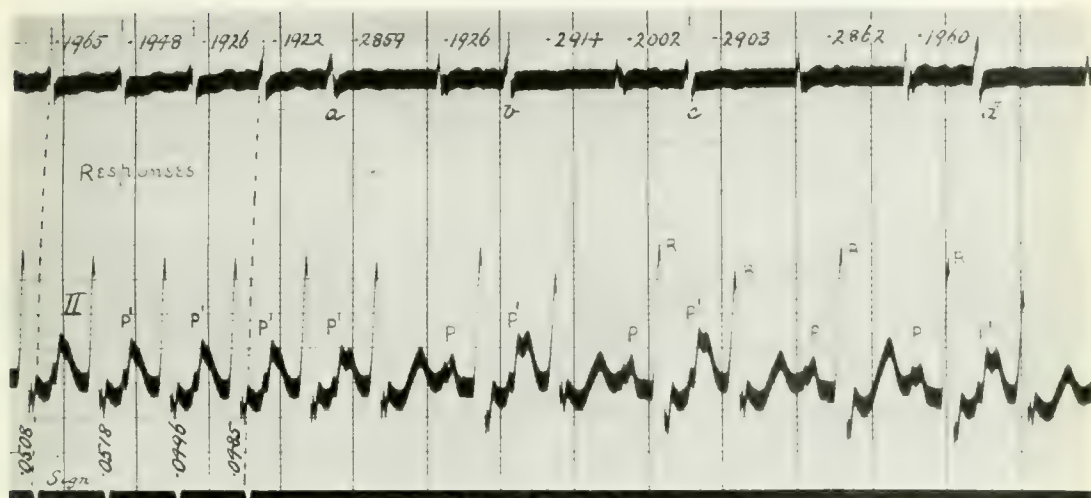


Fig. 18. Dog KD. (Record 5.) Electrogram taken from the mid-caval region (Z contact above) and an electrocardiogram from lead II, showing the after-effects of rhythmic stimulation of the right appendix. A single beat (a) follows the end of stimulation and then the normal rhythm, interrupted by premature beats (b, c and d) retrains. The figures, written vertically, are the intervals between signal marks and intrinsic deflections.

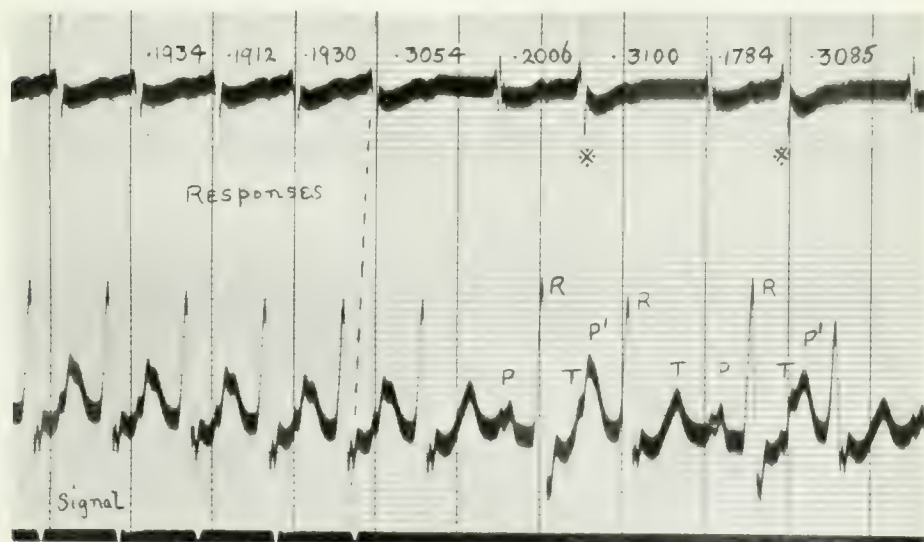


Fig. 19. Dog KD. (Record 3.) A similar record, taken in the same manner, showing a bigeminal action at the return of the normal rhythm.

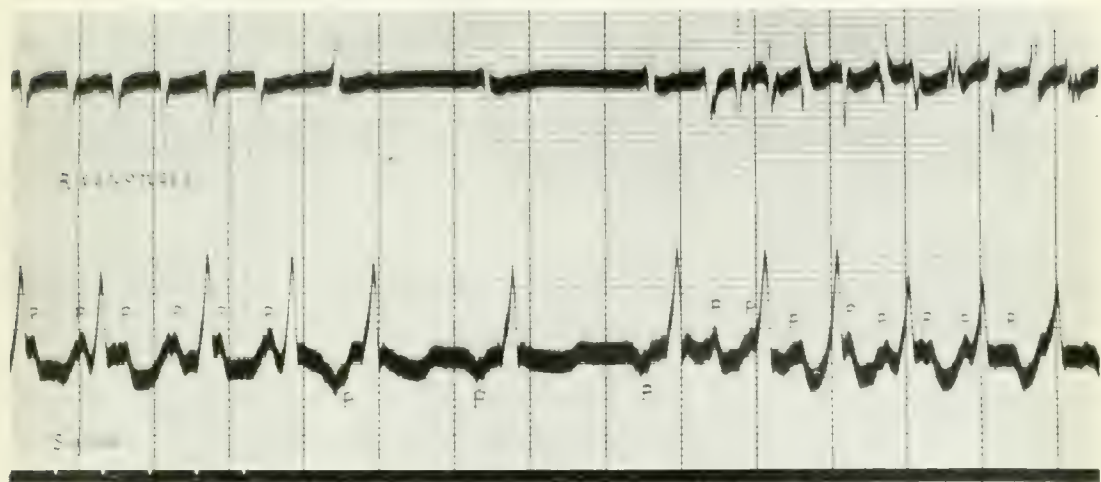


Fig. 20. *Dog KP. (Record 16.)* Electrogram from the upper part of tentorium (Z contact below) and an electrocardiogram from lead II, showing the after effects of stimulating the mid-caval vein rhythmically. A slow physiological rhythm (from some new focus) succeeds the end of stimulation and then the auricle passes spontaneously into impure flutter.

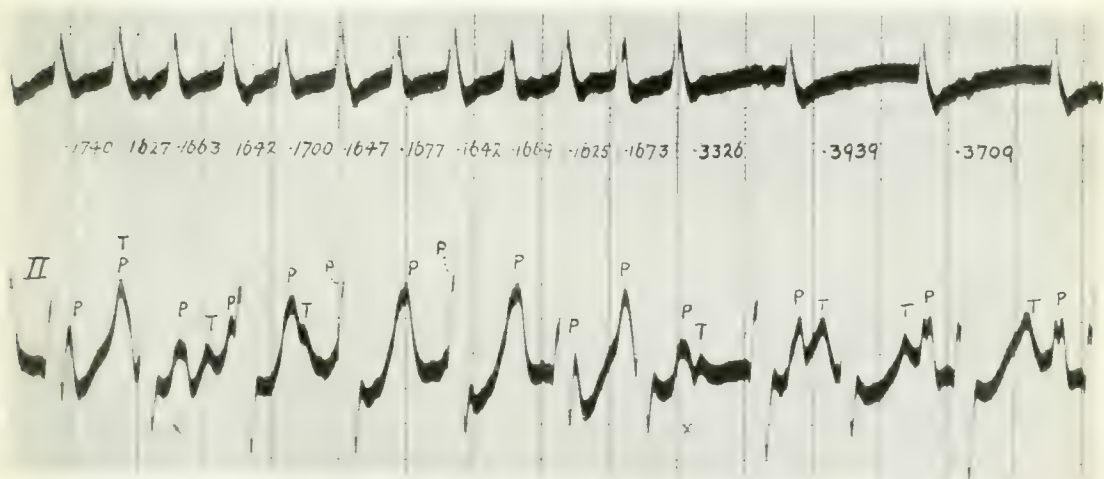


Fig. 21. *Dog KP. (Record 25.)* Electrogram from a mid-caval lead (Z contact below) and an electrocardiogram from lead II. The record shows the end of a period of flutter, induced by stimulating the inferior vena cava rhythmically.

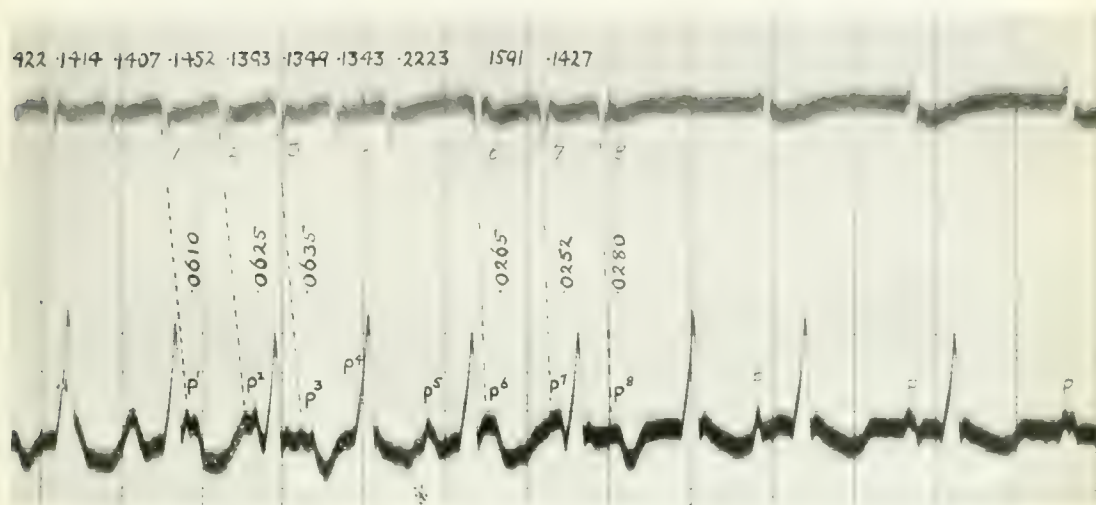


FIG. 22. *Duck No. 13, April 24.* Electrogram taken from the base of the right appendix, near the vena Z contact below, and an electrocardiogram from lead II. The record shows the natural ending of a period of flutter lasting 1 minute and 10 seconds, induced by rhythmic stimulation of the inferior vena cava. The figures written vertically represent the intervals between auricular complexes and corresponding intrinsic deflections.

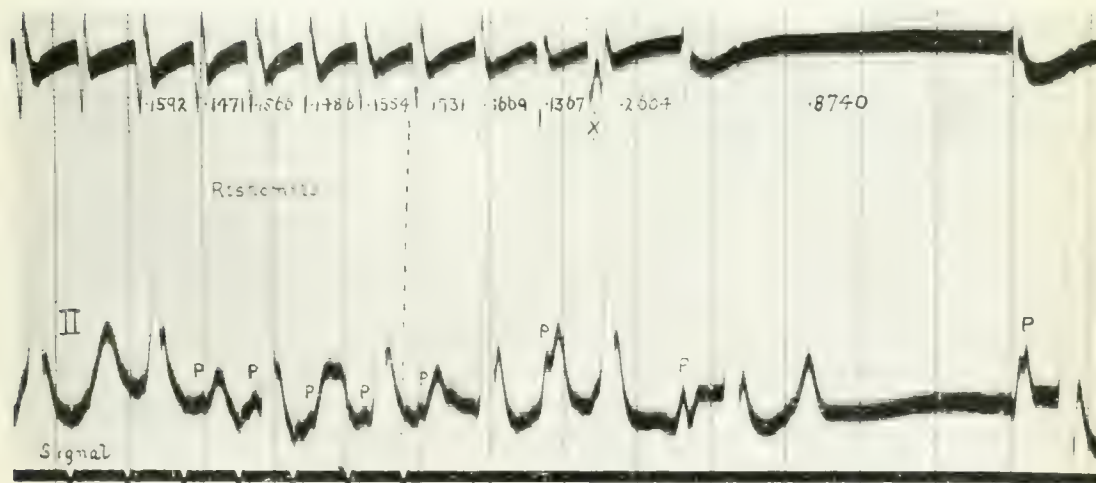


FIG. 23. *Duck No. 13, April 2.* Electrogram taken from a mid-caval lead, Z contact above, and an electrocardiogram from lead II. The record shows a short after effect of stimulating the inferior vena cava rhythmically.

OBSERVATIONS UPON FLUTTER AND FIBRILLATION.

BY THOMAS LEWIS.*

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PART IV.—IMPURE FLUTTER; THEORY OF CIRCUS MOVEMENT.

IN a previous article a condition produced experimentally in the auricle is described, to which it seems desirable to restrict the term auricular flutter. In this condition the auricle beats rapidly and its cycles follow each other with a high degree of regularity, as electrocardiograms taken from lead *II* and as direct leads from the auricle demonstrate. Another feature of the condition, no less distinctive than the last and associated with it, is the orderly character of the events in the auricle. In all regions of the auricle examined, the excitation wave as it regularly crosses the contacts produces deflections which are uniform in amplitude and form in successive cycles, an observation which shows that the direction taken by the excitation wave in relation to any pair of contacts is constant from cycle to cycle. The cycle in pure flutter unquestionably comprises events which are accurately repeated in the succeeding cycles: pure flutter is an orderly affair, and this statement applies not only to the body of the auricle but to all parts of the muscle, including that of the appendages and that forming sleeves to the great veins. From further observation it appears that this orderly sequence and repetition of the auricular movement depends upon the excitation wave travelling in a circular fashion in the body of the auricle and at a uniform though depressed speed, and upon the centrifugal spread of the excitation wave from this circle to those parts of the auricular tissue which, though they do not lie in the actual circle, are in union with it. We may speak of the single wave in flutter as consisting of two parts, the *central or mother wave*, and the *centrifugal wave*, which proceeds from the border of the central wave and courses over outlying muscle.

Now, disturbances of the normal auricular rhythm which are brought about by rhythmic stimulation of the auricle, and which persist after such stimulation is withdrawn, are not always of this kind: they vary from the type, in which the movements though abnormal are quite orderly, to types in which disorder is of very great complexity. Between these extremes are

* Working on behalf of the Medical Research Council.

TABLE SUMMARISING OBSERVATIONS ON DOG KQ.

1ST AFTER-EFFECT. This resulted from a single tap of the right appendix with the handle of a forceps it lasted 14½ minutes. The auricle had passed for a short while into flutter spontaneously when the anaesthesia was being opened.

Record No.	Minutes after onset	Muscle region examined.	Interventricular deflections (electrogram)	Auricular complexes (Lead I I).	Auricular rate in (Lead I I).	
1	0.3	Mid-caval	Irregular	Disturbed	494	
2	4.3	<i>I. V. C.</i>	Alternating	Disturbed	470	Fig. 14

2ND AFTER-EFFECT. This was produced by stimulating the mid-caval region with rhythmic induction shocks at a rate of 437 per minute. It lasted 26½ minutes. Pure flutter developed at record 9, when the rate fell.

3	2	Base of right appendix	Irregular	Disturbed	500	
4	3	Mid-caval	505	Fig. 13
5	5.7	<i>I. V. C.</i>	500	
6	6.5	<i>I. V. groove</i>	507	
7	8.5	<i>S. V. C.</i>	Regular	..	497	Fig. 10
8	10	Tip of right appendix	Irregular	..	498	
9	14.5	<i>I. V. C.</i>	Regular	Regular	448	
10	17.5	Mid-caval	458	Pure flutter
11	19.9	<i>I. V. C.</i> (low)	449	
12	21.3	Base of right appendix	458	
13	22.5	Tip of right appendix	Slightly irregular	Slightly disturbed	453	
14	23.7	<i>S. A. N.</i> region	..	Regular	452	Fig. 7
15	25	<i>S. V. C.</i>	..	Slightly disturbed	448	Fig. 8

3RD AFTER-EFFECT. This resulted from rhythmic stimulation of the *I. V. C.* at a rate of 476 per minute. It lasted 3 minutes.

21	0	<i>S. A. N.</i> region	Irregular	Disturbed	479	Fig. 16 of Part III.
22	1.5	..	Regular	Regular	448	Fig. 17 of Part III.

4TH AFTER-EFFECT. This resulted from rhythmic stimulation of the *I. V. C.* at a rate of 521 per minute. It lasted 1½ minutes.

23	0.9	<i>S. A. N.</i> region	Regular	Regular	411	
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5TH AFTER-EFFECT. This resulted from rhythmic stimulation of the mid-caval region at a rate of 573 per minute. It lasted 35 minutes, and has been fully described in Part II of the present series of articles.

29 to 41	5.1 to 33.3	Many surface areas	Regular	Regular	368 to 380	Pure flutter
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transitional types, some of which being relatively simple can be analysed, some of which being less simple permit of partial analysis. These transitional types, simple and more complex, are the subject of the present article.

Auricular flutter presenting simple disturbances.

Description naturally begins with the very simplest examples. Several of the curves now to be described were taken from an animal, in which a period of pure flutter, lasting some 35 minutes, was examined in detail in Part II (5th after-effect of the accompanying table). The period of flutter in question serves as one of the chief illustrations of the thesis previously presented, that flutter is essentially a circus movement. But, before this period of pure flutter was induced, after-effects, some short and some long, were obtained, in which the events were somewhat different. The first curves used as illustrations were taken at different stages of an after-effect lasting some $26\frac{1}{2}$ minutes (the second after-effect of the accompanying table). The after-effect was produced by rhythmic stimulation at a rate of 437 per minute. During the middle period of this after-effect (Records 9-12), a period of about 10 minutes, pure flutter was exhibited; but at the beginning and at the end of the after-effect the flutter was impure.

Fig. 7, the first example, was taken when the after-effect had lasted $23\frac{2}{3}$ minutes; below is a curve from lead *II* and above an electrogram from paired contacts on the base of the right appendix near the tænia, the *Z* contact lying towards the body of the right auricle. The curve from lead *II* is not to be distinguished from a curve of pure flutter. The auricular complexes present double summits, which are evenly distributed throughout the whole curve. In the electrogram from the base of the right appendix the deflections are uniform in sequence, form, and amplitude, if we accept four points of the curve. At four points (*a*, *b*, *c* and *d*) the curve is disturbed; otherwise it would form a perfect example of a pure flutter curve. The disturbance consists of a striking change in the form and amplitude of the deflections. The chief or intrinsic deflection, for most cycles, is written as a very steep and thin line which runs off the top of the figure and indicates that for these cycles the excitation wave passed up the tænia and appendix: the unusual deflections are of much less amplitude, *a* and *b* starting as short upstrokes, *c* and *d* as short downstrokes. All these deflections are premature, the first three by about a hundredth of a second, the last by a few thousandths of a second. *All are compensated*, none produces a disturbance of the general and regular action in the mass of the auricular tissue. If the evidence that pure flutter is dependent upon a circus movement is accepted, it must be acknowledged that a circus movement is in progress in this auricle and that this circus movement is undisturbed. That the disturbance, such as it is, is a local one and that the main movement in the auricle proceeds in its usual fashion, is clearly shown by the shape of the auricular complexes in

lead *II*, corresponding to the deflections *b* and *d*; these remain unchanged: and the relation of the intrinsic deflections to the auricular complexes is precisely the same at the beginning and end of the record. How is the disturbance to be explained? There are alternatives. The excitation wave, which the unusual deflection signals, is an offshoot of the mother wave or it is not. In the first circumstance the disturbance is due to local aberration of the excitation wave from its accustomed path for a single cycle, in which case the disturbance is the result of altered conduction; in the second circumstance it is due to a new impulse arising in the heart and is an extrasystolic phenomenon. Either explanation, and no other explanation seems open to us, would sufficiently account for the phenomenon. The effects of fresh excitation waves, started in an auricle during flutter of that chamber, have not been tested sufficiently, but, accepting the conclusion that flutter is due to a circus movement, much would seem to depend upon where the new impulse entered the muscle. If it entered at some point of the vital central circle we should expect that it would bring the flutter to an end or that it would deflect the central wave from its circuit and alter the length of that auricular cycle;* if it entered, not the circle, but a part of the muscle supplied by the centrifugal wave, a disturbance of the central wave would not necessarily follow, for the new (centripetal) excitation wave might meet the centrifugal wave and be brought to a standstill. The other explanation assumes altered conduction,† and would confine this alteration to a limited area of muscle passed over by the centrifugal wave. Such a change of conduction could not be supposed to occur upon the path of the central wave, for, if it so occurred, it would in greater or lesser degree upset the central wave; but if conduction altered on the path of the centrifugal wave, the central circuit would not be influenced, the change would be confined to a non-vital part of the outlying muscle.

Thus, we arrive at this general argument; assume a circus movement in flutter, and it is necessary to assume that, so long as the rhythm of that flutter shows no derangement—as indicated by the regular incidence of its auricular complexes and the return of the intrinsic deflections to their compensatory positions—the irregular arrival of excitation waves at any point of the surface under examination is due to changes confined to muscle lying outside the range of the central wave.‡

Of our two explanations, each of which suffices to explain premature beats in the electrogram, neither can for the moment be given preference.

* The cycle would be prolonged the more according to the prematurity of the new excitation wave; for the more premature the latter the greater would be the area of muscle involved by it.

† In this instance an alteration in the nature of improvement, the supposition being that by the removal of a barrier the wave finds a shorter path to the contacts

‡ It is, of course, conceivable that there is a certain degree of option for the central wave; it might happen that two closely adjacent and similar paths lead it to the same point in the same, or almost exactly the same, period of time. In these circumstances the one path might be blocked without producing any perceptible change in the time relations of the circuit.

Regard the question from the opposite standpoint and inquire what are the expected changes, when new impulse on the one hand and altered conduction on the other come into play. The first will produce premature beats. The second will produce premature beats when the conduction improves: it will produce delayed beats when conduction becomes more depressed.

The next figure (Fig. 8) is from the same period of flutter and was taken a little more than a minute after the curve last described; the electrogram was taken from the superior vena cava, well above the tænia, but inside the reflection of the pericardium, the contacts lying approximately in the line of the vessel with the Z contact below. The first disturbance of an otherwise regular flutter comes at *a*. With the exception of this deflection, the intrinsic deflections of the curve indicate movement of the excitation waves up the vessel, against the direction of the blood stream. The deflection in question is premature: the inter-intrinsic interval which follows it is prolonged.* The example is very similar to the last considered. But in the same curve are two deflections which are distinguished from the rest by their greater amplitude and by the greater depth of the dips which precede and succeed them; both these deflections are delayed, both are followed by inter-intrinsic intervals which are of unusual shortness. The delay in the appearance of these deflections is compensated.

In other instances, the unusual form of deflection is neither premature nor late, but falls at the point anticipated. Fig. 9 is an example of this kind from another animal. This comes from a period of disturbed flutter lasting 4 minutes and 55 seconds, and resulting from rhythmic stimulation of the inferior vena cava. In lead *II*, the auricular complexes occur throughout the curve at regular intervals, and approximately twice as frequently as the ventricular complexes. The regularity of the electrogram is disturbed by the deflections *a* to *f*, which, though falling at anticipated points, are of unusual form. In examples of this kind, as in those of the previous curve, an extra-systolic origin will hardly serve as an explanation. A slight alteration in the course of the wave as it approaches the contact will.

Examples of such simple disturbances as are here described are comparatively rare; usually the changes are more complex. Of simple disturbances in our collection there are more instances of prematurity than of delay in the unusual deflections; the departure in form also appears to be greater in degree for premature than for delayed deflections amongst our examples. Whether these facts are significant cannot be stated, as the number of curves is not large. Although conduction changes sufficiently account for the disturbances described, it is not always possible clearly to conceive the courses of the corresponding excitation waves relative to the contacts. For example, in Fig. 8, it is not obvious why a wave (disturbance

* The short and long cycle together last 0.2651 of a second: the average length of the two is 0.1325 of a second. This is a little lower than would be expected if compensation were perfect (*i.e.*, 0.1340 of a second); but the actual divergence is trifling, and some allowance must be made for error in measurement.

a) which reaches the upper contact (of the superior cava) first should be premature as compared with the waves reaching the lower contact first; *a priori* a longer course might be anticipated* and the beat would then be delayed. This difficulty probably arises out of our having no information as to the precise course of the waves, and no certain knowledge of the course of the central wave in these instances.

When the electrograms are disturbed, it is not possible to map out the course of the central wave, for the following reasons. Readings from different points show inconsistencies, and readings repeated from the same point often show material divergences. When flutter is disturbed, the rate of conduction on the centrifugal path is probably lower than in pure flutter and is less constant; although the speed and course of the central wave often show no change from cycle to cycle, as in the examples so far described, yet change in the path of the central wave is evidenced by other records. Thus, in Figs. 7 and 8 the rate of the basal rhythm is such as to yield cycles of about 0.1340 of a second in duration; these curves were taken after the after-effect had been in progress for 23½ and 25 minutes respectively. Fig. 10 was taken when the same after-effect had lasted 8½ minutes and the length of the cycle is about 0.1200 of a second. Not only is there this change in the rate of the basal rhythm (for other examples of which the table may be consulted), but there is evidence of change in the direction of the central wave; the general form of the auricular waves in lead *II* shows a notable difference in Figs. 7 and 10; the conspicuous notching of the complexes in Fig. 7 is lost.

In attempting to map out the course of the central wave, in after-effects of this kind, the change in rate of the basal rhythm in itself introduces an error, though one which is probably not serious. But material changes in the form of the auricular complexes render accurate plotting impossible; the common standard to which all measurements from different direct leads referred is not maintained.

It is the rule, in dealing with after-effects consisting of disturbed or impure flutter, to find that, although for short stretches the basal rhythm shows no disturbance, yet from minute to minute it changes to a greater or lesser extent in rate and other characters.

Disturbances of a less simple kind.

In the examples of solitary changes in the form of the intrinsic deflections described, it is to be noticed that the auricular complexes in lead *II* are of distinctive form and maintain their outlines and time relations with considerable exactitude from cycle to cycle throughout the period of disturbance. Upon this observation it is concluded that these examples of

* Though it is not difficult to conceive that the course might be shorter, as later examples will demonstrate.

impure flutter consists essentially of the same form of central circulating wave as exists in pure flutter, and that the disturbance, such as it is, is a local phenomenon and is confined to the path of the centrifugal wave. For if the course of the wave varied over any considerable proportion of the auricular tissue, assuredly the form of the auricular complex would change correspondingly. The constancy of form from cycle to cycle is in itself sufficient evidence that the course of the central wave is constant, for the central wave guides the excitation over the greater part of the auricle.

Now when we come to more complex disturbances, this constancy in the form of the auricular complexes in lead *II* is no longer maintained, though in many curves the skeleton of these complexes is clearly to be recognised. To illustrate this point two curves are published: these were taken from a single animal (Figs. 11 and 12). Each figure shows an electrocardiogram from lead *II* and a simultaneous curve taken by a direct lead from the inferior vena cava. The first of these curves (Fig. 11) is from a long period of pure flutter, described in detail in a previous article. In lead *II* there is a series of regular auricular complexes, uniform in amplitude and form. Corresponding with this is a series of intrinsic deflections in the direct lead: these deflections are regular in incidence, in amplitude and in form, and they possess constant time relations to the auricular complexes.* Compare this figure with Fig. 12, taken from a short after-effect produced by faradic stimulation of the right appendix (as was the after effect which Fig. 11 illustrates). In this figure the actual termination of the after-effect is shown. In lead *II* we recognise at once a series of auricular complexes, each of which mounts to a bifid summit. Whereas in Fig. 11 the measured intervals between auricular summits varies by no more than 5 thousandths of a second, the corresponding intervals in Fig. 12 vary by 19 thousandths of a second. There is a further difference: the auricular complexes of Fig. 11 are uniform in shape, those of Fig. 12 are not. There are slight but quite distinct changes from cycle to cycle: note especially the inconstancy of the notches (marked X) which precede the main upstrokes. Now these are not artefacts, for when the after effect ends the curve becomes perfectly smooth: they represent actual events in the auricle. When we turn our attention to the direct lead the cause is manifest. The events in the region of the inferior cava are complex, far too complex to analyse fully. There is, however, a series of little sharp peaks, to which dotted lines are drawn, occurring at fairly regular intervals and clearly related to the auricular summits in lead *II* (the intervals between the latter and the former are written vertically on the curve). In addition, there are deflections marked by a small circle: these usually (not always) succeed the sharp peaks referred to and at an interval which varies. In the earliest part of the same curve there are deflections of a quite different kind (see asterisk). Now although a complete interpretation

* The intervals from the summits of the auricular complexes to the beginning of the intrinsic deflections are given below the electrogram from the inferior cava.

of this figure is impossible, yet it may be interpreted in part. To explain it we suppose that a circus movement is in progress in the auricle, and that this is constantly repeated, with but little change in its course or time relations; the movement in the main mass of the auricular tissue is maintained with sufficient constancy, and for that reason the skeleton of regular auricular complexes is clearly inscribed in lead *II*. It is supposed that in a considerable mass of the auricular tissue the course of the excitation wave is varying, varying in a complex way, and that this mass includes the region of the inferior cava; the tissue involved is of sufficient extent to affect materially the axial electrocardiogram, so that the skeleton curve in lead *II* is clothed and in part hidden by minor deflections of the string responding to this disorder. It is not thought probable that the irregular spaced auricular complexes in lead *II* represent disturbance of the central circuit, but that the disorder in the muscle on the centrifugal path distorts the electrical representations of the central excitation wave in lead *II* and that in consequence equivalent points in the measurement of these cycles are not found. This conclusion is come to because the degree in which the cycles in lead *II* vary in length is not great, and distortion of the complexes and the error which it introduces into the measurements seem sufficiently to account for the variation.

Now it is the rule, though there are occasional exceptions, to find that where the disorder is great in the direct lead, the auricular complexes of lead *II* are more or less distorted in form and arrangement (see Table on page 294); and from examples such as that now described we pass by transition to others in which the skeleton in lead *II* is no longer recognisable. When we come to these last we come to curves which clinically would be termed records of auricular fibrillation. The last subject will not be pursued further for the moment.

To return, it must be obvious that if the view held in regard to Fig. 12 is a correct view and that there are disorders of the auricle in which, while the central wave progresses regularly, large outlying masses of muscle are excited in a disorderly fashion, it would happen from time to time that the direct lead would lie on muscle belonging to the track of the central wave or supplied regularly from it. Briefly, we should expect to obtain figures in which the partly hidden skeleton of the auricular complexes appeared in lead *II*, while a perfectly regular series of deflections was yielded by the direct lead. Of this Fig. 10 is a probable example: such curves are not very frequent. The picture displayed by lead *II* is barely recognisable as one in which flutter plays the predominant part. Yet the general sweep of the complexes, alternate ones coinciding with ventricular deflections, is there; individual complexes are, however, much distorted. Corresponding to each of these distorted complexes is a steep upward deflection in the direct lead: these deflections comprise a series, regular in form, amplitude and spacing. The curve was taken from the superior vena cava (the *Z* contact being towards the body of the auricle). The record is explained by assuming that the superior cava, and the muscle path lying between it and that involved

by the central wave is conducting uniformly, while in some other considerable part of the centrifugal path conduction is varying. Thus, there are disturbances which display themselves in lead *II*, while given curves of direct leads fail to indicate them: there are disturbances which appear in direct leads, but which are insufficient materially to influence the electrocardiograms from lead *II*. Usually, however, if the local disturbance is at all complex, both curves display it. These observations are readily explained if we assume local variations in conduction.

An example which shows simple disturbance locally, and in which the auricular complexes of lead *II* are not inconsiderably affected, is shown in Fig. 13. This curve was taken in the early stages of an after-effect, several curves from which have been described (see Table on page 294): the after-effect consisted in part of pure and in part of impure flutter (Fig. 7, 8 and 10). The auricular complexes in lead *II* are ill-defined: there are approximately two to each ventricular cycle, but they have suffered a good deal of distortion. Corresponding to each auricular complex is a large downward deflection in the electrogram, which was taken from a mid caval lead (with the *Z* contact below). The downward deflections show the excitation wave to be travelling down the tania for the corresponding cycles. There are, however, three deflections (*a*, *b* and *d*), which show that the wave passed in a direction almost at right angles to the contacts, while two others (*c* and *e*) indicate a movement of the excitation wave up the tania. In addition to these relatively gross disturbances, another form of interference is displayed. When the deflections are regular in amplitude and downwardly directed, the intervals between them alternate in length, slightly but distinctly. The first disturbance (*a*) falls precisely at a time when a deflection is expected. The second disturbance (*b*) is slightly premature, the interval which precedes it is shorter than usual (0.1097 of a second). A short interval, but not so short an interval as this, is expected if the alternation of length is to continue. Then follows the third disturbance (*c*); this is also preceded by a shorter interval than usual (0.1138 of a second), which is more noteworthy in that it is now the turn for a relatively long cycle. A long cycle follows (0.1327 of a second), which exactly compensates the whole disturbance and the events are then repeated. It is of interest to note that throughout the curve the basic rhythm is not affected: this is shown by the constancy in the length of the groups of cycles which are indicated by dotted brackets.

An example of continuous local alternation in the lengths of cycles accompanied by alternation in the heights of deflections is seen in Fig. 14. This curve was taken from the inferior cava (the *Z* contact being above) and comes from a period of disturbed flutter induced by a single sharp tap of the auricular appendix with the handle of a knife. The impure flutter lasted $4\frac{1}{2}$ minutes, and the present curve was taken shortly before it ended. This type of alternation resembles in every respect that which is seen during rhythmic stimulation of the auricle at high speed, where the auricle is responding to each stimulus, and where the alternation can scarcely be

caused other than by local change in conduction; it is naturally attributed to the same cause in the present instance.

An instance of alternation in the amplitude of deflections, in the absence of recognisable alternation in the lengths of cycles—a phenomenon also witnessed in curves where the auricle is responding to rapid rhythmic stimulation—is seen in Fig. 15. This is from a 20 second after-effect of stimulation (*S. A. V.* region), the lead being from the mid-caval region (*Z* contact above). In this record, there is in addition a single disturbance of another kind, the direct lead showing a single upright spike, which is very slightly premature and which is subsequently compensated. The auricular complexes of lead *II* are recognised in the centre of the curve by means of their bifid summits, lying a little more than 0.02 of a second apart. Alternate complexes fall at the beginning of ventricular systole, but in these the two summits may still be identified: the second falls with the upstroke of *R* as indicated in the figure. In the early part of the same curve and again near its ending the auricular complexes change a little in form, the second summit being less distinct, but if we use the first summit for purposes of measurement, the intrinsic deflections of the top curve are found to precede it by constant intervals of about 0.02 of a second, with the exception of the interval between the slightly premature intrinsic deflection and the corresponding auricular summit in lead *II*; for this cycle the interval widens to 0.029 of a second.

To sum up: there are after-effects in which auricular flutter appears as the basal condition, but in which there are local disturbances of a relatively simple kind. These disturbances are compensated, that is to say, they do not interfere with the basal rhythm of the auricle: sooner or later, responses to the main movement are perfectly restored. The disturbances may be attributed to one of two causes, namely, the origin of new impulses on the one hand, or to local changes in conduction, on the other. The evidence favours the second mode of production. First, because those excitation waves which approach the contacts in an unusual direction are not always premature: sometimes they form a perfect sequence with the remainder, sometimes they are delayed. Secondly, because certain of the disturbances clearly resemble those which are witnessed when the auricle is responding to rhythmic stimuli sent into it at high speed, in which circumstances an extrasystolic origin is scarcely to be considered. And thirdly, because the rates at which the auricular muscle is beating when these impure forms of flutter prevail is very high, higher than those prevailing in pure flutter in the same animals (see Table on page 294): the rates are similar to those which are known to strain conduction in the auricle, producing obvious disturbances of rhythm, when the question is tested by response to rhythmic stimulation. It is concluded that local changes of conduction are alone responsible. As pure flutter is regarded as due essentially to a circus movement so also are these examples of impure flutter, it being concluded that the local changes are confined to the centrifugal path of the excitation wave, and that they do not affect, or do not appreciably affect, the movement of the central wave.

Complex disturbances.

A few selected curves are now described in which the events are of a high grade of complexity, but in which useful analysis is possible. An attempt to analyse disorders of the heart beat which at the very start are known to be extremely complex is unlikely to prove successful, unless we are prepared to consider detail. Such an analysis has led to what is believed to be a correct interpretation of a number of curves, which at first sight appeared hopelessly involved. It is to be emphasised that if we are certain that the current which gives our record is derived solely from the heart, then every movement of the string, however small, has a meaning, which if read, will take us a little nearer to understanding the disorder as a whole. It is only by precise measurement and remeasurement, and by careful charting of many of the curves, that the nature of these comes partially or wholly to light.

First example. The first example of impure flutter (Fig. 16) arose as an after-effect of faradic stimulation of the inferior vena cava. The figure presents a curve from lead *II* and an electrogram from the right auricular appendix (*Z* contact towards the body of the auricle). The after-effect lasted 15 seconds and the record represents its middle phase. Examining first the curve of lead *II*, we recognise that it is essentially a flutter curve. The auricular complexes are disposed regularly throughout the curve, but in the early phases they blend alternately with the ventricular complexes. At the end of the curve, the relation of the ventricular beats alters a little and successive complexes (18-24) become perfectly distinct. All the distinct auricular summits of the curve have been numbered. Numbers 3, 5, 7, 9, 11, 13, 15 and 17 fall within the ventricular summits and fail to appear distinctly; but we may be sure that they are there: traces of them are actually visible and the corresponding ventricular summits are raised in amplitude. The curves of this figure have been accurately measured and charted in Fig. 1 (page 317), to which we may now turn. The auricular complexes are charted below as vertical lines (*A.C.*); the unbroken lines represent summits actually measured, the broken lines represent those which are calculated by dividing the distance between adjacent summits. As the auricular complexes in the last part of the curve occur at almost exactly equal distances from each other, this method of procedure is justified and introduces no material error. By charting we are able to reconstruct the auricular portion of the curve and to show that while adjacent cycles are of almost exactly the same length, there is lowering of auricular rate as the curve proceeds: it takes place somewhat abruptly in the neighbourhood of the 12th auricular complex. Now the auricular complexes throughout the curve are very similar in form, and it is to be concluded that whatever has happened, the central wave has pursued the same or a very similar circular course throughout the whole period recorded. There has been some change, however, for the auricular rate slows down. To what may this change be

attributed? To one of two events. The rate of conduction may have fallen slightly in the auricle as a whole, thereby lengthening the time taken to complete the circuit; or a local change may have happened, the track which the central wave travels may have become obstructed at one point and the wave have been deflected slightly out of its course, its course thus being prolonged. The latter explanation is the more probable, since the slowing is relatively abrupt; the change of rate is a jump from a higher to a lower level; it is not gradual. But it is not the meaning of the changed rate which is now of chief interest, it is what simultaneously happens under the contacts placed upon the appendix. The deflections *m* to *r* (Fig. 16) are of constant form and are of similar amplitude; they follow each other with a high grade of regularity (the limits of the inter-intrinsic intervals were actually 0.1394 and 0.1319 of a second, and these extremes form the two first beats of the series), and bear an almost constant relation to the summits in lead *II*. The stretch of curve is identical with what is seen in pure flutter* which is continuous. But the intrinsic deflections of the first half of the curve (*a* to *j*) present no such order; they are irregular in form and in amplitude, they occur at intervals which vary by as much as 5 hundredths of a second. From deflections *e* to *j* there is an alternation both in the height of the deflections and in the length of intervals separating them. That does not constitute the whole change, there is further and obvious irregularity; a conspicuous change in the position of the deflections relative to the summits of the auricular complexes exists (Fig. 1): whereas, in the later stretches of the curve, an intrinsic deflection follows each summit after an approximate interval of 0.05 of a second, deflections *e* to *j* fall almost simultaneously with the summits, and from *a* to *e* there is a gradual change. The curve may be divided into two periods, each of which presents a regular action of the main mass of the auricle: the rate is not precisely the same in the two periods, but otherwise the action of the auricle is continuous. In the later and slower period, pure flutter prevails; in the earlier and faster period there is a local disturbance of a complex kind. The change in rate, on the one hand, and the re-arrangement of the intrinsic deflections, on the other, are simultaneous, and, in offering an explanation, it is impossible to dissociate the two. In the light of previous observations, the conclusion seems irresistible that the disorder of the intrinsic deflections in the first half of the curve is consequent upon the higher rate which then prevails. These observations in themselves suggest the cause: for we know that if a portion of the auricular tissue is made to respond to very high rates of stimulation, its conduction is at first generally depressed and that a similar depression of conduction exists in pure flutter; we know that if the rate of stimulation is further advanced, local changes of conduction are apt to occur; we may assume that the same event is likely to happen when in pure flutter the rate becomes raised. It is to be regretted that the curve does not record the beginning

* It is not quite pure; alternation is present.

of the disturbance as well as its ending;* we must content ourselves therefore by showing that the curve as it stands is interpretable on the basis of changed conduction. The interpretation adopted is displayed in the figure. The central wave is circulating in the auricles, and each time it circulates it throws off a centrifugal wave into the right appendix. Thus, each circus movement, represented by the summits 13 to 22, yields a response in the appendix after an almost constant interval. To circus movement 10 there is no response, to 11 there is the response *k*, the interval being shortened because of the preceding rest in the appendix; the next interval (12-1) is longer, and from here on to the end of the curve the intervals, though of almost the same length, show slight alternation (which is reflected in the lengths of the corresponding inter-intrinsic intervals). The events which precede circus movement 10, which is supposed to yield no response in the appendix, are compatible with a gradual increase of the intervals (between the passage of the central wave and the response of the appendix) up to the point of the block. Thus, if we introduce the local factor of changing conduction we are able to explain the events of the whole curve. During the faster circus movement conduction in the centrifugal path is strained, and the centrifugal wave passes slowly to the appendix; the difficulty of its passage increases until it is obstructed for one cycle. During this period of increasing obstruction, conduction tends to alternate, thus producing a further disturbance of the responses. Once a centrifugal wave becomes blocked, conduction is freer and the next wave (11-1) is transmitted more quickly, the following wave (12-1) is transmitted more slowly; no doubt the events of the first part of the curve would be repeated, but at this moment the rate of the circus movement becomes slower. From this point onwards the responses are all relatively quick and the intervals of response are constant except that the tendency to alternate is maintained.

It is of interest to note, in support of this view, the precise point at which block comes. It comes at the end of a period of lengthened intervals; but these intervals, while lengthening, also alternate; the block comes in sequence with the long interval (see cycles 4, 6, 8 and 10).

Another point deserves notice. In dealing with the simpler disturbances of flutter, it was argued that such could be explained either on the basis of new impulse formation or on the basis of conduction changes. The same line of argument may be applied to the present curve. Can this curve represent flutter, disturbed locally by a series of new impulses? The answer is hardly in doubt, seeing that the lengths of the inter-intrinsic intervals are in the average a good deal higher than are the lengths of the basic cycles (.10-.11) in the disturbed stretch of curve. Response of the appendix to new impulses, following each other at a greater rate than those of the basic rhythm, is conceivable; but response of one part of the auricle to impulses of slower rate than those

* That could be done only by the use of films as it is impossible to measure the curve on the paper. The objection to films is that they do not permit of the same accurate measurement.

governing the main mass of tissue is a phenomenon of which we have no knowledge: it only becomes conceivable if we suppose that the two masses of auricular tissue are separated by an area of depressed conduction. It cannot be conceived that the appendix responds more slowly than the main mass (as it obviously does in the present illustration) unless depressed conduction in the tissue between the appendix and the body of the auricle is admitted. In other words, we may be sure that the curve is, in part at least, the result of disturbed conduction. If the impurity of the flutter is explainable on the hypothesis of conduction changes only, and conduction changes are known to be in part accountable for it, the only warrantable conclusion is that these changes are wholly responsible.

Second example. Fig. 17 shows an electrocardiogram taken from lead *II* and an electrogram from the superior vena cava. The pair of contacts was so placed that the *Z* contact lay on the sulcus terminalis and the *C* contact on the superior vena cava above it, the two lying in the line of the superior vena cava. The record is of an after-effect resulting from stimulation of the inferior vena cava and lasting 5 minutes: the record was taken 1 minute before the after-effect ended. The electrocardiogram exhibits ventricular deflections *R* and *S* and a regular series of auricular complexes (1, 2, 3, etc.) which fall in varying relationship to the ventricular elements of the curve. As in the last example, some of the auricular complexes are partially concealed by the ventricular deflections: the regular sequence is distinct towards the end of the curve. The complexes have a uniform general outline, but are slightly deformed by small notches and little alterations of contour here and there; some of the more conspicuous of these are marked by small asterisks. Now these notches and deformities are not accidental, for control curves obtained from this animal, which, like all our animals, was under deep anaesthesia, were perfectly smooth. Each such small notch has a meaning significant of the mechanism which we are studying, whether or not that meaning is at the moment apparent. Associated with these slight deformities of an electrocardiogram, which otherwise would be considered compatible with a condition of pure flutter, is an irregular electrogram. Where the auricular complexes are deformed in curves from lead *II*, it is the rule to find an irregular electrogram; there are exceptions to this rule, one of which we have noticed (Fig. 10), in which the electrogram is supposed to have been taken from the path of the central wave. Real deformity of the auricular complexes in lead *II* may always be read as indicating local disturbances. To simplify description the curves are charted in Fig. 2. In this chart the intrinsic deflections (*E.W.*), representing the excitation waves, are plotted to a base line, as vertical lines drawn up or down to correspond with the curve itself: they are labelled alphabetically, *a* to *o*, and the intervals between them are shown by the figures written horizontally immediately above the base line. Below this the auricular complexes are plotted (*A.C.*) as vertical lines, and these are numbered 1 to 17 immediately below the vertical lines; each complex

was plotted by measurement, with the exception of complexes 2, 4, 6, 8, 10 and 14: these last have been filled in by dividing the gaps in which they occur. The lengths of the auricular cycles, as measured from the first and lowest depression of each complex, are expressed by figures written horizontally and placed between the corresponding vertical lines. The degree of regularity expressed by these last readings is of a high order: it is indeed of a similar order to that found in pure flutter, and, taken in conjunction with the *almost* uniform shape of the complexes in lead *II*, speaks clearly for a uniform movement in the main mass of auricular tissue. The rate of this movement is unvarying throughout the curve.

Now consider the electrogram. The main deflections (*a*, *b*, *c*, etc.) unquestionably represent active changes beneath the contacts: they are abrupt deflections representing considerable potential differences: they are the intrinsic deflections, each of which represents the arrival of an excitation wave in the muscle beneath the contacts: but these excitation waves do not reach the contacts rhythmically, they are placed irregularly. Moreover, they do not always first reach the same individual contact of the pair, sometimes the *Z* contact (upward deflection), sometimes the *C* contact (downward deflection) is first reached. Thus, although in so far as the main mass of the auricle is concerned, the electrocardiogram indicates an orderly repetition of events, the electrogram, signalling the local events in the superior vena cava, shows that in this region there is no uniformity. The question again arises, to what extent is the disordered movement in the caval region dependent upon or independent of the movement in the main mass? Are any or all of the deflections in the electrogram produced by spread of centrifugal waves from the regularly passing central wave: or do these deflections represent excitation waves of different and entirely independent origin? If we look closely at the electrogram, certain minute deflections appear upon it. I have marked them (1, 2, 3, etc.) on the curve. These are unidirectional but minute. They occur at *regular* intervals and fall in varying relations to the chief deflections of the curve. If we examine the relation of these minute deflections to the auricular complexes in lead *II*, it at once transpires that they are associated with the main and regular excitation wave which is travelling in the body of the auricle. Now because these deflections are minute, it may be said that they do not represent the arrival of excitation waves actually *beneath* the contacts, but in the neighbourhood of the contacts only:* they are minute extrinsic effects. In other words, the central wave, or a regularly coursing centrifugal wave, comes near to, but does not actually encounter, the superior caval contacts. It comes to the vicinity of the superior caval contacts at regular intervals, as is shown by the uniform relation of the minute deflections to the auricular

* The alternative interpretation that they are minute because both minutes have been reached almost simultaneously is not tenable for several reasons, of which the chief is that they do not show the sharp multiple phases as occur in such circumstances.

complexes in lead *II*. These little deflections are charted as vertical lines in Fig. 2, immediately above and to the right of the corresponding auricular complexes: the figures standing to the left of these lines and written vertically represent the measured (or in a few instances calculated) intervals between the auricular complexes in lead *II* and the little deflections in the electrogram. These intervals are almost constant throughout the chart, such differences as occur being accounted for probably by error in measurement. The small deflections (Nos. 1 to 8) are in a rhythmic series: the 9th small deflection is not visible: it is replaced by the tall intrinsic deflection *h*. This intrinsic *h* is the tallest in the curve, and it represents a movement up the cava in, or almost in, the line of the contacts. For this cycle the wave has passed in the direction in which the centrifugal wave is accustomed to pass, when pure flutter prevails. Consider the intervals between auricular complexes and the succeeding intrinsic deflections: these intervals are written horizontally along the top of the figure (*AC-EW*). The tall intrinsic *h* follows an auricular complex by the shortest interval of any intrinsic deflection in the curve. This fact, taken in conjunction with the form and height of the deflection, indicates that the path of the centrifugal wave has for this cycle been shortest. Further examination lends strong support to this view: for a distinct relation exists between the direction and amplitude of the intrinsic deflections and their time relations to the auricular complexes. Thus, the time relations of deflections *b* and *c* are not dissimilar to those of *i* and *j*: all these deflections are upright. The time relations of deflections *d* and *e* are similar to those of *k* and *l*, and these deflections are all downwardly directed. The upright deflections are preceded by the shortest intervals (*AC-EW*), the downward deflections by the longest: the path which an excitation wave must take to reach the *C* contact first (downward deflection) is longer than that which it must take to reach the *Z* contact first (upward deflection). Regard the intrinsic deflections as a whole and they show a series of transitions in form, marked by the dotted line which runs throughout the chart.

The intervals rise gradually throughout the curve in two stages. Interpret the whole curve in terms of disturbed conduction. The rate at which the intrinsic deflections succeed each other is slower than the rhythm in the mass of the auricle: this fact in itself proves disturbed conduction as it did in the case of the last example of impure flutter. Regard the longest inter-intrinsic intervals (0.2559 and 0.2288 of a second) as the intervals during which the wave fails altogether to reach the superior cava, and correlate this with the gradual increase of the intervals before and after these long pauses, and with the direction of the deflections and the mechanism begins to be clear. The intervals 2-*b* and 9-*h* are shortest and the path to the superior cava straightest (yielding upright deflections), because on each occasion there has been rest.

An interpretation, based on these observations, is given below the figures. A numbered circle represents the central wave in the auricle, pursuing its

course in the same fashion at each cycle. The contacts (*C*, white, and *Z* black) on the cava are represented above it, and the centrifugal wave is shown as leaving the circle and taking its course to the contacts. This course is represented as varying. On its course the centrifugal wave usually passes the *Z* contact and, in passing it, gives rise to the little deflection (*N*), to which attention has been drawn already: if the wave fails to pass the *Z* contact, but strikes it by a short and direct path, the intrinsic deflection is upright and the little deflection (*N*) is replaced. If the wave travels past the *Z* contact without striking it and by a circuitous or sinuous course reaches the *C* contact first, it produces a little deflection (*N*), it subsequently produces after a long interval a downward intrinsic deflection. It is not intended, by means of this diagram, to suggest the precise course of the circuitous or sinuous wave, of that there is no certain knowledge: the diagram is intended simply to show that the curve can be analysed completely on the basis of disturbed conduction. Thus, there is no reason to conclude that the intrinsic deflections of the caval lead represent excitation waves travelling from an independent source: on the contrary, the evidence strongly points to their being directly dependent upon the regular central waves, and that the change in amplitude, direction and position (relative to the last waves) is brought about by changes in the course pursued from the body of the auricle to the contacts and by occasional failure to reach these contacts.

Third example. This presents some points of resemblance to the second example: in some degree it resembles the first example: it also has points of its own. The example consists of two records (Figs. 18 and 19) taken within a few minutes of each other from the same animal.* Each corresponds to a separate after-effect, produced by faradising the appendix: the first lasted 5 and the second lasted 7 seconds. In each record the lower curve is an electrocardiogram from lead *II*, and the top record is from the body of the right auricle, the *C* contact lying towards the inferior cava and the *Z* contact towards the base of the appendix. The two records, when analysed, prove so closely alike in the main features that there can be no doubt the mechanism was fundamentally the same when each was taken.

These two records represent a not uncommon variety of after effect: as in the present instance, such an after-effect is usually transitory: it is an unstable mechanism. It is of considerable consequence, in that it represents the last of a series of transitions between pure flutter and mechanisms hitherto spoken of as auricular fibrillation. The present mechanism is the last of the series, in the sense that it is the most complex which appears to permit of detailed analysis.

Taking the simpler record first, namely, Fig. 18, we see in lead *II* a series of tall and irregularly placed ventricular deflections *R*. In addition,

* Two further curves taken under the same conditions from this auricle show the same essential features.

the curve presents a series of summits, labelled 1, 2, 3, 4, etc. These summits occur at approximately equal distances from each other; they are for the most part fluid, some (such as 1, 8, 9 and 12) conspicuously so; but they vary a good deal in detailed form. Were there a little more deformity than actually exists, anything like accurate measurement of the summits would not be possible.* Were the deformity a little greater; were it as great throughout, for example, as in the vicinity of complex 6, the curve would almost pass as an illustration of auricular fibrillation.

In the early part of the electrogram is a series of upright intrinsic deflections *a, b, c*, etc., which vary in amplitude, slightly in form and in spacing; in the last part of the curve the deflections are downwardly or upwardly directed, and the spaces between them are cut up by a series of smaller deflections. This record has been charted in Fig. 3; and it is to be stated that this chart, like others of its kind, have been constructed with every recognised precaution as regards accuracy. The curves have been repeatedly remeasured and checked so that there may be as little chance of error as possible. The auricular complexes (4, C) were actually measured from the second little peak on the summit; but this point has not been charted; the point charted is the upstroke of the auricular complex, computed by allowing a constant interval between upstroke and peak.† The intrinsic deflections, representing the arrival of the excitation wave beneath the contacts laid on the auricle, have been charted above. The rule adopted in charting these deflections is to chart the beginning of the sharp upstroke or the sharp downstroke, as the case may be. The intervals between successive auricular complexes and between successive intrinsic deflections are written horizontally in decimal points of a second. It is to be emphasised that in considering these figures, due allowance is to be made for error of measurement. The error where the intrinsic deflections are concerned amounts probably in no instance to more than a thousandth of a second; where the auricular complex is concerned the error is larger, though it probably lies well below a hundredth of a second in most instances. The basis of the last statement will appear in a moment. At the end of Fig. 18 the electrogram is disturbed by a series of small deflections: the numbered deflections (7, 10, 11, 12, etc.) stand in almost rhythmic series and are clearly related to the corresponding auricular complexes. They are charted as vertical lines in Fig. 3 (N) and the intervals which separate them from the corresponding auricular complexes are given in decimal points of a second

* It is only possible to do this by the exercise of careful discrimination.

† This point has been chosen to avoid trouble by the charting of this record, and that next described, inasmuch as the peaks present the most constant points for measurement; in the next record the upstrokes do. The average distance between the two points has been ascertained, in itself a useful check to the measurements, and allowed in charting. Actually, in the case of any given record, the interpretation is unaffected by the point of the auricular complex chosen as the standard for measurement, providing that corresponding points are used for all cycles of the curve.

‡ A few auricular complexes in this chart have been filled in by dividing the gaps in which they lie; these are expressed in dotted lines (as in Figs. 1 and 2).

below the chart (*AC-N*). These intervals are very similar, approaching for the most part closely to zero. The little numbered deflections are of the same origin as those discussed in the second example of impure flutter: they are extrinsic deflections and are due to the passage of the central wave (or a centrifugal wave from the latter) near to the contacts without actually encountering them. The value of these little waves to us in the present instance is great, for they form from time to time a most useful check to the accuracy with which the auricular complexes are charted. The two series of deflections (*AC* and *N*) fall with such constant relations to each other (see also Fig. 4), that it is beyond doubt that they owe their origin to a common cause. It may be that the intervals between corresponding members of the two series should not be *precisely* the same; but the degree of their uniformity gives a clear idea of the maximal error in charting the auricular complexes. The relationship suggests that the error is likely to be greatest in so far as complexes 15 to 19 are concerned; reference to the original figure will show that it is precisely these complexes in which the second peak of the bifid summit is least defined; these complexes vary most from the standard form, and measurement, as a consequence, becomes less accurate. Thus it becomes apparent that such errors as occur in the charting of the auricular complexes are sufficiently small to be immaterial. We come to the interpretation of the record.

This record differs from those previously described in that the intervals between adjacent auricular complexes vary materially. The series has lost that precise rhythm which is so characteristic of pure flutter and of examples of impure flutter previously described. The actual variation amounts to as much as 0.037 of a second (Fig. 3), and is clearly to be made out in the original curve (Fig. 18). Thus complexes 3 and 4 are obviously further apart than are complexes 11 and 12. There may be a circus movement in progress in the auricle: there almost certainly is; but it is not a precisely uniform circus; there is some variation of the path, or some variation of speed in the path, from cycle to cycle.* The variation in the lengths of the cycles is most conspicuous in the early part of the curve; it disappears at complex 13 for five cycles, *and these cycles are short*. For these last cycles the time in which the main circuit is completed is lessened, and with this lessening it tends to become uniform. The quicker circuits start between complexes 9 and 10; the lengths alternate for four cycles and then settle down.

It is to be seen in the charted electrogram that in the early period of the record each intrinsic deflection stands in relation to an auricular complex. Conceive the circus movement as a wave travelling around the great caval veins and passing up the tænia. Outside the actual circuit a corresponding centrifugal wave will travel through the body of the right auricle and passing upwards will strike the *Z* contact of our direct lead.†

* The first is the more probable, since the changes are abrupt.

† It will be remembered that the lead was from the body of the right auricle, the *Z* contact being towards the inferior cava.

But the events of our curve at this stage are not quite orderly. There is some variation in the circuit itself, as displayed by the irregular spacing of the auricular complexes: there is probably a further slight irregularity in conduction from the circuit to the muscle area examined. The last path varies a little, and, as a consequence, the intervals ($AC-EW$) vary a little and the amplitude of the intrinsic deflections varies. The two phenomena go hand in hand, the greatest variation in interval ($\beta - c = 0.0212$ of a second) is associated with the smallest intrinsic deflection: but throughout the period of curve covered by complexes *I* to *9* or *10*, the variation of intervals is insufficient to suggest serious disturbance of the centrifugal path. It is at this point (complex *9* to *10*) that an improved state of conduction* happens in the main circuit, which soon leads, as has been seen, to the steadier flow of the central wave. This improved conduction, dependent presumably upon the removal of a small barrier on the shortest course of the central wave, quickens the circus movement, and the centrifugal impulses, passing out from the circus more frequently, throw a heavier strain on conduction in outlying muscle. As a result, the speed of conduction in this outlying muscle falls, the path becomes more variable and the intrinsic curves begin to assume a more irregular appearance. The intervals ($AC-EW$) increase steadily, and gross changes in the form of the intrinsic deflections are witnessed before long. Here, then, is a second and equally striking example of what we have already observed in the instance of Fig. 1. According to our hypothesis of impure flutter, the basal phenomenon is a circus movement. Speaking generally, this circus movement is steady in the early phases of such disturbance, which is confined to outlying muscle on the centrifugal path. But there is this curious relation: if at any time the path of the main circus movement is deflected by a small local barrier to its progress, its path becomes a little longer; when the path becomes longer the completion of the circuit is delayed, the rate of the circus movement is retarded, the centrifugal impulses are correspondingly less frequent. As these centrifugal impulses become fewer, defects of conduction on the centrifugal path become less evident, for this path is less strained. Conversely, when the local barrier on the path of the central wave is removed, that path becomes shorter; the circuit is more quickly completed: the rate of movement is increased; centrifugal impulses are thrown off with correspondingly increased frequency, and the local disorder grows.

To return to our present record. We have seen that with the shortening of the intervals between auricular complexes, the intervals between the latter and the intrinsic deflections grow. They grow from a value of 0.0148 to 0.1325 of a second. Associated with this lengthening the intrinsic

* In using the term improved conduction, it is not intended to convey that there has been an improved function of the auricle as a whole; the improvement is supposed to be a local one; whether this is to be regarded as an increased power of the original track to conduct, or whether it is to be regarded as the removal of a local obstruction, shortening the circuit, the term "improved conduction" is applicable. The last hypothesis is strongly favoured.

deflections change their form, indicating that the centrifugal wave approaches the contacts by a new path. For two, and perhaps three, cycles (l , m and n)* the C contact is first encountered; later, although the path becomes longer, the Z contact is first encountered (n' , o , p , etc.); but although the intrinsic deflections become upright again, their original form (as Fig. 18 shows) is not resumed, the upstroke (and especially the downstroke from the summit) is less steep; the form of the intrinsic curves is now peculiar, a fact which will be referred to again. At the end of the record, when conduction to the contacts is at its slowest, the auricular complexes widen their distances again (to 0.16165 of a second), but they do not widen early enough to prevent one wave (19) from being blocked in its course to the contacts on the auricle. As a result of the rest, which the tissue under the contacts thus obtains, and in part perhaps as a result of slowing in the main circuit, the next wave (20-s) goes through.† It goes through to the contacts in much the same time as do the early waves of this record, and the original form of the intrinsic deflection (as Fig. 18 shows) is restored.

This record confirms the interpretation of the first examples of impure flutter described, and strongly supports the contention that impure flutter is dependent on variations in conduction, confined in some instances to the centrifugal path, in others appearing to affect both centrifugal and central paths.

The second record from this animal (Fig. 19) is in many respects similar to that just described. It is charted in Fig. 4. We may notice, first of all, the resemblance between the two charts. The present chart (Fig. 4) shows throughout disturbances similar to that found over the last half of the chart already discussed (Fig. 3). The rate of the basal rhythm (expressed by the $AC-AC$) intervals is the same over the whole of the present chart, as it was over the latter half of the last record. This sameness of rate confirms the view previously expressed, that the disturbance in the muscle on the centrifugal path is brought about by the receipt of very frequent impulses, these embarrassing conduction in the muscle. If we compare that portion of Fig. 4 which lies between auricular complexes 7 and 11 with that portion of Fig. 3, which lies between complexes 16 to 20, a striking resemblance is at once seen and this resemblance is found to extend to fine details. Thus, the corresponding $AC-AC$ and $AC-EW$ intervals are of almost the same lengths in the two charts: the last show progressive increase of length until in both instances a figure 0.132 of a second is reached; following upon this, there is in both instances an unusually long inter intrinsic interval, interpreted as due to the blocking of a centrifugal wave. The next $AC-EW$ interval is conspicuously short‡ in each chart. Compare the amplitudes of deflections

* The meaning of the curious double deflection (n , n') will be discussed at a later stage.

† The exact relation between the wide $AC-AC$ intervals and the block is not quite clear; the subject will be discussed again at a later stage.

‡ A negative value (-0.0066 of a second) has no special meaning, the readings are relative and not absolute. Absolute readings are not obtainable in that there is no real beginning to an auricular complex; one continues into the next.

g , h , and i in Fig. 4, with the deflections p , q and r in Fig. 3; they are very similar; and the form of the deflections is similar, as a comparison of Figs. 18 and 19 will show. The one stretch of chart and curve is almost a replica of the other.* Further, the intrinsic deflection s (Figs. 3 and 18) which is associated with the short $AC-EW$ interval following the supposed block, is similar in form and amplitude to intrinsic deflection j (Figs. 4 and 19), which has similar associations. Briefly, it is beyond doubt that the two stretches of chart and curve represent identical mechanisms. Take Fig. 19 by itself (and the corresponding chart, Fig. 4) and compare the first stretch leading up to deflection i , with the stretch leading up to deflection s . Except in minor detail and in the number of cycles over which the changes occur, the one stretch is a repetition of the other. Take the last half of Fig. 3 and compare it with the two similar stretches in Fig. 4; and the conclusion becomes inevitable that the disorder in the auricle is of a phasic kind, repeating itself as it proceeds. A further important conclusion comes from this exact periodic repetition of detail; it shows quite clearly that the positions of the intrinsic deflections are controlled by the positions of the auricular complexes, or that the first and the last are under common control. These curves place out of court the interference of excitation waves of independent origin. That the impurity of some forms of auricular flutter is not dependent on new and independent impulses is a conclusion already reached from an examination of simpler forms of disturbance; the present records place that conclusion on a firm basis. It is a conclusion of paramount importance, for we are left with the alternative which ascribes the impurities of flutter to changes of conduction.

A curious disturbance in these two records remains to be described. This description will complete the analysis of the records, and will be significant from two other standpoints; it will link these curves with the more complex disturbances seen when the actual responses of the auricle to very rapid rhythmic stimulation are recorded (see the third article of this series and especially Fig. 6 of that article); it will help to link the disorderly movement

*There is another resemblance, which is probably not without significance. The longest $AC-EW$ interval in each chart is followed by a similar lengthening of the $AC-AC$ intervals (to 0.16165 and 0.16935 of a second, respectively); the occurrence of lengthening at this particular moment in a second record, points to the lengthening being something more than accidental at this time, though I am unable clearly to see the reason of it. It looks almost as though the lowered conduction (resulting from increased rate and exhibited by the $AC-EW$ intervals) was in some way responsible for the change. It might be suggested that when conduction becomes most depressed in the centrifugal paths and the centrifugal waves are taking their most unusual and circuitous courses, that one such wave might find its way by an unusual channel back into the central track. But, though this is an evident possibility, it is extremely hypothetical, and the case for it is somewhat weakened by some uncertainty in regard to the lengths of these particular $AC-AC$ intervals. That lengthening of the intervals occurs is beyond doubt, but it is not certain where it commences. There is in each chart a space, which when divided gives two intervals of unusual (but equal) length. The first long intervals are calculated to be 0.16165 and 0.16935 of a second, respectively; but the intervals between the corresponding small deflections and these are probably the more reliable—are 0.1552 and 0.1390 of a second, respectively. In view of the uncertainty, the discussion cannot profitably be carried further. It is, however, to be observed that such uncertainty as exists does not invalidate the main interpretation of the charts, as this is given in the text. The broad features of the curves remain and are explained, although this particular feature remains somewhat obscure.

of the auricle underlying the present records, with those obtained when the auricle fibrillates (as will be shown in a further article). The disturbance consists of the two oppositely directed but paired deflections, first a downward and then an upward deflection), a single example of which is seen in Fig. 18 (n, n'), and many examples of which are seen in Fig. 19 (c, c', d, d' , etc.). The disturbance in a pronounced form is seen in Fig. 19. If the paired deflections c, c', d, d', e, e' and f, f' are followed as they succeed each other, it will be noticed that the deflections forming the pairs separate as the curve proceeds. Precisely the same thing happens with the second series m, m', n, n', o, o' in the same figure. But what is of further interest and consequence is the manner in which this pairing of opposed deflections begins and ends. Take the downwardly directed deflections by themselves: as they succeed each other they form a diminishing series which fades to nothing: they begin in series with previous and simple downwardly directed deflections, which with a solitary exception are of greater amplitude. Take the upwardly directed series: as these proceed they form a series which augments in amplitude: beginning in nothing: they increase in size, until, where the pairing ends, they continue as a series of simple upward deflections of increased amplitude. The explanation of these curious changes is to be found in the time-relation of the downstrokes (of these paired deflections) to the auricular complexes. The downstrokes follow the upstrokes of the auricular complexes by very constant intervals, varying (in Figs. 18 and 19) by no more than 0.0146 of a second (the limits of variation are 0.0216 and 0.0362 of a second). If we exclude a doubtful example (namely, Fig. 19 m, m'), the variation is no greater than 0.0073 of a second. The downstrokes of these paired deflections are more directly related to the central waves in time than are the upstrokes of the pairs: or to put the matter in another way, the increasing delay in the transmission of the excitation waves to the direct contacts is not shown by these downward deflections, as it is by their upright companions. The downward deflections are interpreted in the following manner. Take the series of downward deflections b, c, d, e, f , of Fig. 19. The deflection b is of considerable amplitude: such have already been interpreted as intrinsic deflections representing excitation waves which strike the C contact (as opposed to the Z contact) of the pair first of all. Now, if we so interpret b it is difficult to avoid interpreting the deflection c similarly, and if c , then d, e, f . But sooner or later we are driven to accept the alternative deflections c', d', e' or f' , for these grow in prominence. That two excitation waves coming from different directions meet in each instance under the contacts is hardly conceivable, for in that case the individual deflections of each pair should stand in a more constant and close time-relation to each other. The alternative explanation is that at first the excitation waves approach the contacts from the direction of the C contact, but that as the series proceeds, these excitation waves approach less and less closely to the contacts, while the excitation waves coming towards the Z contact enroach more and more. The barrier on the side of the C contact becomes increasingly wide; that is the

explanation. There is no need to divorce the individual waves of the pairs, when we consider their origin. The waves c and c' (or d and d' , etc.) are twins of the same mother wave. When the excitation wave finds its channel to the C contact blocked it pursues its course, and this course brings it sooner or later to the contact Z , but, in approaching or in passing the contacts in its effort to reach C it leaves a greater or lesser impression upon the contacts; briefly, the downward deflections begin as intrinsic deflections: they become, more and more, as the series progresses, extrinsic deflections. Now this explanation harmonises with the time relations of the deflections. The excitation wave approaches the C contact up to a certain point in the muscle without unusual difficulty; and at an almost constant time interval, relative to the auricular complex, it leaves its impress on these contacts; but as the barrier increases in width, this impress becomes smaller; simultaneously, the wave after reaching a barrier which is increasing in width, cycle by cycle, is deflected through a longer and longer course before it can reach Z ; so it happens that the interval between the twin waves, and between the auricular waves and the upright deflection of the pair, increases. Apply the same reasoning to the upright deflections: in their earliest phases these are extrinsic deflections, in their later stages they are intrinsic deflections, ultimately the excitation wave sweeps across the contacts completely from the Z side, as it did in the beginning from the C side. Thus, one set of deflections gradually replaces the other. From time to time it will happen, maybe, that the two branches of the excitation wave will reach their corresponding contacts *almost* simultaneously; in such circumstances, so it is imagined, the curiously composite waves (illustrated by m , m' , Fig. 19) are brought about.

The transitions as a whole bring another supporting evidence of *sinuous* movement in the auricular wall, as one result of failing or overstrained conduction. It has been repeatedly suggested, and much evidence has been brought forward to show that in auricles in which artificial stimulation produces disorderly movement, as opposed to the orderly movement of pure flutter, that such disorder results from local variations in conduction; it has been suggested further, and it is again emphasised, that when disorderly action prevails, the disorder is not attributable so much to a local fall of conduction rate, but to the setting up of local obstructions or actual barriers, islands or strips of tissue, through which progress is delayed or is for one cycle barred.

Finally, it is pointed out that, as the after-effects of stimulation, when analysed, are found to be of varying degrees of complexity, leading up from pure flutter, by easy transitions to complex examples of impure flutter in which eventually the basal or circus rhythm shows clear signs of breaking up, so we may look for a further form of disorder in which the simple circus movement is lost and the auricle is invaded by re-entering waves, following sinuous and changing paths. From these preliminary and *a priori* considerations we are led to ask if this may not be the underlying mechanism in that most complex of all the after-effects of stimulation, namely, auricular fibrillation. This hypothesis will be examined

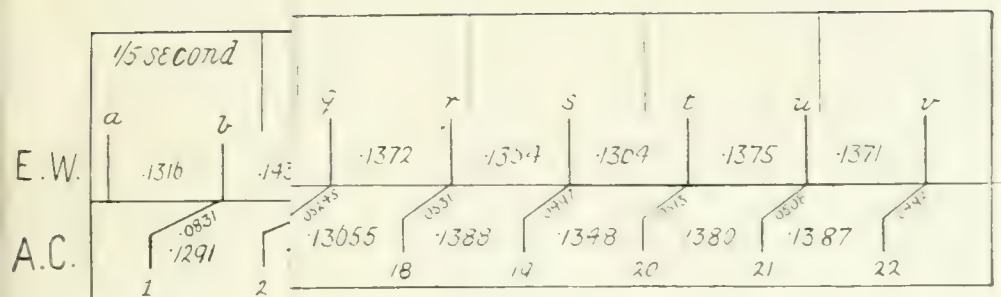


Fig. 1. A chart comparing the summit of auricular lead with that of Fig. 16. The inter-

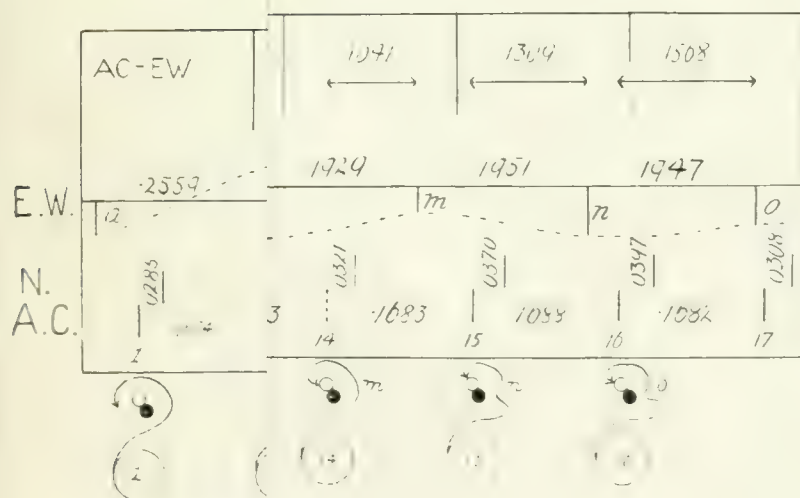
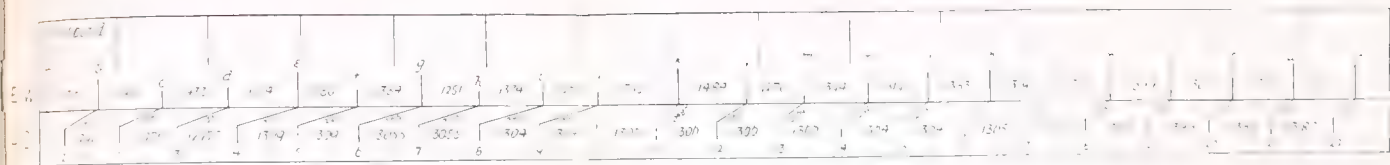
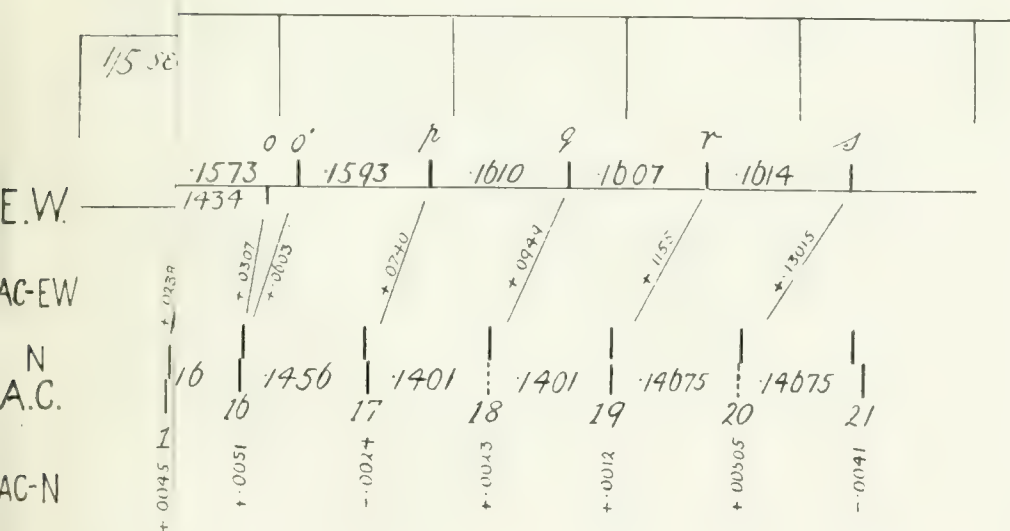
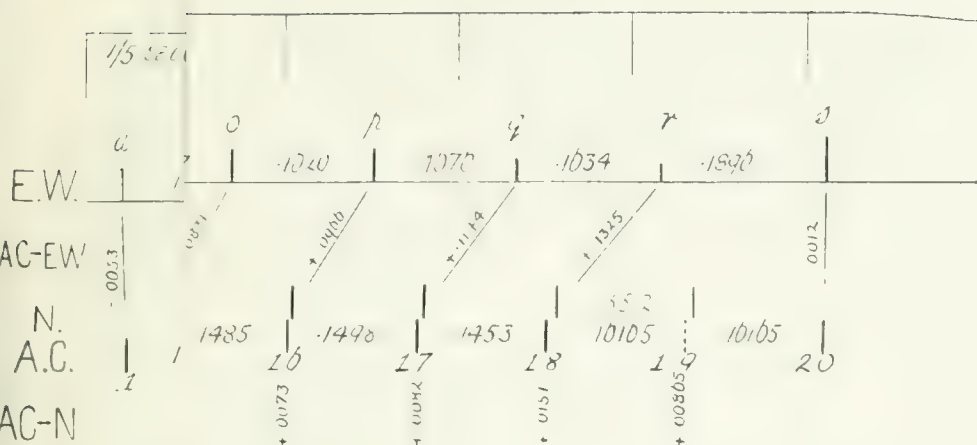
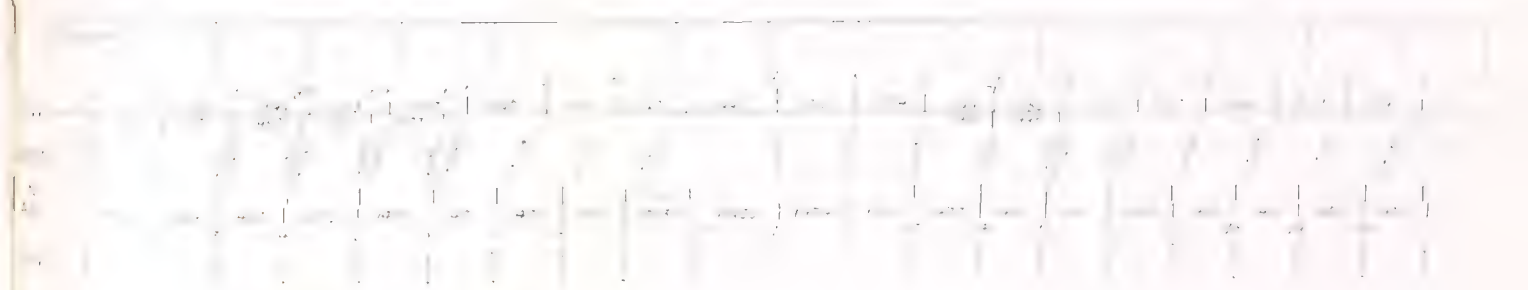


Fig. 2. A chart comparing the point of auricular lead. The fig. deflections of complexes and of the excitati-





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in a later article. Meanwhile it should be clearly stated that the hypothesis relating to fibrillation of the auricles, which is reached by the present observations, is similar to that which has been expressed by Mines³ and by Garrey,¹ who argue from quite distinct data. It would not be convenient here to discuss these observations, those of writers upon the same lines, or their hypothesis at length. Greater justice can be done to these in describing observations upon fibrillation itself; present-day theories of the cause and nature of this disturbance can then be discussed more adequately.

THE THEORY OF CIRCUS MOVEMENT IN THE AURICLE.

The description of the experimental work, in the present series of articles, has now taken us sufficiently far to discuss the theory of circus movement in the auricle as the underlying mechanism of flutter in more detail. Mines' experiment upon a ring of muscle has already been quoted in a previous article. The events which follow when a ring of muscle is stimulated, whether that ring is excised or whether it is a natural ring or cylinder, may be illustrated by means of a series of diagrams. Suppose that a ring of muscle is stimulated by means of a single induction shock, at *a*, the centre of its lowest quadrant (Fig. 5, 1). The muscle responds and a wave of excitation (and contraction) flows from the point stimulated in both directions through the ring. The wave moves uniformly along the two sides of the circle and eventually its two borders meet at the centre of the upper quadrant (Fig. 5, 4). In these diagrams that portion of the circle of muscle which in contracting has entered the refractory state is blackened. When the advancing borders of the wave of contraction meet, the whole muscle has become involved: if the refractory state lasts, as it is supposed to do in these diagrams, for a greater time than it takes the wave to travel from *a* in the lower quadrant to *b* in the upper quadrant, the whole of the muscle ring is refractory, and is for the moment incapable of responding to any impulse which reaches it. This state of complete refractoriness lasts for a certain period, obviously depending upon the duration of the refractory state at a point of the muscle, and upon the time which it takes the contraction wave to pass through the whole muscle. Thus, if the refractory state at a point lasts for a period represented by the numeral 3, and the time taken for the contraction wave to travel from *a* to *b* is represented by the numeral 2, then the period during which the whole circle is refractory will be represented by the numeral 1. If the duration of the refractory state is greater by a certain period of time, at any given point, than the duration of flow of the wave, the whole muscle will be simultaneously refractory by that period of time. When the refractory state passes away it will pass away first where it first began, namely, at point *a* of the ring (Fig. 5, 5), and the state of excitability to stimulation which returns will flow as a wave in the wake of the original wave of contraction. Eventually the whole ring becomes excitable once more (Fig. 5, 8).

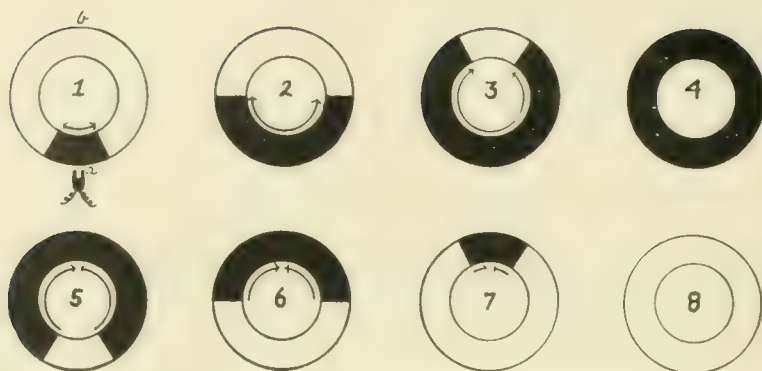


Fig. 5. A diagram illustrating the progress of a single wave passing through a ring of muscle as a result of stimulating it at *a*. The black portion of the ring represents the refractory state, and the figure shows its progress through the ring till it involves the whole (4); later the figure shows its subsidence.

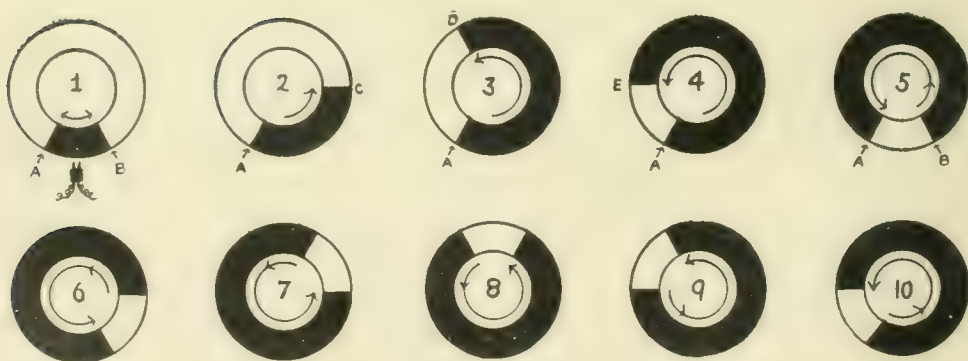


Fig. 6. A diagram to illustrate the establishment of a circus movement in a ring of muscle. The ring is stimulated in its lower quadrant and the wave spreads to *A* and to *B*. At *A* it is blocked, but from *B* it continues around the ring. When it arrives at *E* (4) the refractory state at *A* is passing, and so the wave continues to travel around the circle (5-10).

Events comparable to what is here described are to be distinguished when the auricle is beating normally. The wave passes from the pacemaker, situated upon the ventral aspect of the superior cava, around both sides of the vessel and its travelling borders meet on the dorsal aspect of the cylinder. When two waves of contraction meet they are both brought to a standstill, for each meets a region of refractory muscle. But the auricle is not a simple cylinder of muscle; it has a complex structure, and the wave of contraction flows to all parts of it: the borders of the wave meet in regions other than that described, and the advance is there stayed; in other situations the wave travels to a free edge of muscle, as on the edges of the muscular sleeves of the great veins, and is there brought to a standstill for want of a further muscular path. Thus all parts of the auricular muscle experience contraction, but the wave ceases to progress after a time: the whole chamber enters the refractory state*. When the simple circle of muscle, which we use to simplify description, is stimulated by means of slow rhythmic shocks, the events of Fig. 5 are repeated as each effective impulse enters the muscle. If the rate of stimulation is increased the rate of response is increased and the duration of contraction (and of its accompanying refractory state) becomes decreased. So it happens that if the rate of stimulation is raised gradually a stimulus following a response does not find the muscle refractory, but yields a second response, and so on. A second response may thus be awakened in certain circumstances, before the muscle most distal to stimulation has as yet received the first wave.

But as the rate of stimulation is raised, another event is likely to happen. For a reason, which we shall presently enquire into, a wave of contraction is propagated in *one direction only*. If, when such a wave is propagated, stimulation ceases, there is a curious sequel; the wave proceeds around the circle, and, meeting no wave passing in the opposite direction it returns to the point of stimulation; by this time the muscle has become again excitable at this point, and the wave goes further and repeats its circuit, not once but a great number of times. Now this is Mines' experiment as he described it and much as he explained it, and the course of events in the ring of muscle is illustrated by Fig. 6. The wave of contraction is propagated by stimulation, and involves an area of muscle in the lower quadrant: but while it fails to proceed beyond the point *A* (Fig. 6, 1) it does proceed beyond *B*, *C*, *D* and *E* (Fig. 6, 2 to 5). Up to the moment when it reaches *E*, there has been no progress at *A*, and when *E* is reached, the refractory state begins to subside at *A*. Consequently when the wave reaches *A* (Fig. 6, 5) it finds the muscle in its path excitable and proceeds. The advancing wave follows its own wake (of subsiding refractoriness) and if the advancing border and the wake travel at the same speed, the wave is free to circulate indefinitely (Fig. 6, 4 to 10).

* In the heart of a moderate-sized dog, beating under ordinary experimental conditions, and at rates of 90 to 150 per minute, the length of the refractory state at a point is approximately 0.06 of a second: the time taken for the whole surface to be covered by the contraction wave is a little less than this. The duration of complete refractoriness lasts probably only 1 or 2 hundredths of a second. When the auricle is fluttering the refractory period is certainly much shorter than the figure here given.

Mines knew that the refractory state is shortened by raising the rate of stimulation, and realised that the conduction rate was reduced in the same circumstances: the former would render the first point of the ring stimulated free to respond again after a relatively short interval, the latter would delay the receipt of the impulse. Thus, it would happen that the wave on returning to its original starting point could proceed. Clearly, there would be three factors involved:—

- (a) The length of the muscle path;
- (b) The rate of conduction; and
- (c) The duration of the refractory state.

If the muscle ring were of sufficient circumference, neither a lowered rate of conduction, nor a fall in the refractory state would be essential to the continuance of a circus movement. But very large rings of muscle are not readily obtainable, and to explain the establishment of a circus movement, Mines found it necessary to assume in his rings alterations of the two last cited factors. He was the more disposed to this explanation in that he found that rapid stimulation was required to establish the circus movement, and he knew that raised rate would account for such changes. He assumed that the failure of the wave to pass in both directions was due to a local block, but did not explain how this block is brought about*, neither did he explain why the breaking of rapid rhythmic stimulation sometimes leads to a circus movement and sometimes does not.

Now precisely similar events happen when the auricle is stimulated by rapid rhythmic shocks, in certain circumstances, but the wave circulates around one or other of the natural rings formed in the wall by the inlets of the large vessels or, maybe, by the openings of the auriculo-ventricular orifices: and very similar considerations apply to the auricle as apply to the artificial ring.† It does not matter that these rings of muscle are not

* Mines assumed, as Carey did, the presence of unidirectional block, meaning by this that an impulse can pass in one direction and not in another. Both writers cite observations to support the view that such local and unidirectional block is possible.

† Neither a decrease in the length of the refractory period nor a decrease in the rate at which the wave is propagated is absolutely necessary, theoretically, so it would seem, to the maintenance of the circus movement, which we describe in the auricle around the mouths of the great veins. It has been stated in the footnote on page 323 that the time during which the whole auricle is refractory, when it beats normally at rates of 90 to 150 per minute, is probably no longer than a few hundredths of a second at the most. This statement is based on our recent observations upon the dog's auricle (*Heart*, 1920, VII, 131). The length of the refractory period in the muscle units is estimated at approximately 0.06 of a second, and it takes but a little less than this time for the contraction wave normally to flow over the whole auricle. When the wave travels in a circuit, the circuit is completed approximately in a time of 0.1714 to 0.1034 of a second (the rates in flutter varying between approximately 350 and 580). If the refractory period remained constant in flutter (*i.e.*, if it was 0.06 of a second), the wave would re-enter muscle which had been excitable for periods varying from 0.1114 to 0.0434 of a second. Thus, it is quite unnecessary to postulate a reduced refractory period. That is not to state that such reduction does not occur. Again, if while the heart beats normally, the period during which the whole auricle is refractory lasts at the most a few hundredths of a second, a slight prolongation of the course taken by the contraction wave would be sufficient to produce the condition, in which the last part of the

isolated from the rest of the auricular tissue, for the latter is outlying tissue, and the wave once established in the ring will proceed into these outlying parts and terminate in them. Thus, such outlying parts as the appendices and the sleeves of the veins, or the whole of one auricle when the circus lies in the other, would form outlying tracts and would be supplied centrifugally from the central path. No such outlying part of the muscle can contain so short a path for a re-entrant wave as the central path: if the rate of conduction is uniform throughout the tissue, there is consequently no chance of the wave re-entering from this outlying muscle and interfering with the circus movement. It must be evident that any excitation of the circuit by a wave, other than the circulating wave, would lead to the clashing of the two waves sooner or later, and the circuit would tend to break up and the whole movement to cease. To this argument, however, exception can be taken in one particular, namely, when the supposed circuit flows around the mouths of two vessels (for example, the superior and inferior vena cava): here there is a shorter path, namely, between the two vessels, for muscle fibres separate them. If we find evidence of a circus movement around these two veins, a reason why the bridge separating them is not followed must be found. To this matter we shall return. Meanwhile we may enquire why, as a result of rhythmic stimulation, the wave is ever started in a single direction. It is not a question of the point stimulated. Though it seems true that flutter is more easily propagated from the region of the tænia (stimulation at the upper middle or lower ends of the tænia being most successful), it is also true that it may be propagated from the tip of the right appendix. In point of fact, the path, which will become the central path, is always stimulated, if not directly, then indirectly, through the muscle which intervenes between it and the point stimulated. It would make no difference in the simple experiment of the ring if the stimuli were applied to the ring itself or to a tongue of muscle projecting from the ring. The reason why the wave passes in one direction through the ring is that, as the rate of stimulation is increased, there comes a time, sooner or later, when local block appears, as a result of the strain thrown on the muscle by the rate at which it receives impulses. Consider the circumstances in the light of our experiments on the superior vena cava. We stimulate this vein rhythmically, and, sooner or later,

muscle to be activated contracts after the first part activated has begun to be excitable once more. When the wave passes, as it does in certain experiments, around both cavæ, the course which it takes is a long one. Suppose the wave to be propagated at normal rates around this long path, it is within the bounds of possibility that on some occasions the circus movement might last a longer time than the refractory period at the point of re-entry. Thus, it may not always be essential, though it usually is essential, to postulate a reduced conduction rate. That again is not to state that such reduction is not the rule. But in one of our experiments (*Dog KQ*) the rate of conduction was not very conspicuously less while the flutter prevailed than during the period in which the heart beat normally. Here, however, argument must proceed cautiously. It is possible, indeed it is rather probable, that estimates of normal conduction rates taken in animals anaesthetised with morphia and paraldehyde and from the surfaces of auricles exposed to cooling, are a good deal lower than they should be. It is to be remembered also in the present connection, that the paths in the human heart are longer than in the dog; for that reason, presumably, the rate of flutter in the human heart is less, namely, 200 to 350 per minute.

conduction becomes depressed, and becomes more depressed in one place than another. When we stimulate the auricle we are stimulating the ring of tissue which ultimately is to become the path of the central wave; as the rate of stimulation is raised the conduction rate begins to fall. As we have seen, in auricles predisposed to flutter, this fall is uniform or fairly so; but even in such auricles when the rate of stimulation rises high enough local effects are witnessed. A local block* in the central path will account for the wave becoming confined to a single direction in the central path, just as it will account for it in the excised ring of muscle. It is a noteworthy fact that flutter in the auricle is started by rhythmic stimulation whose rate usually exceeds, by a good deal, the rate of the subsequent flutter; for in pure flutter itself conduction is uniformly depressed and local blocks are not maintained at the rate prevailing. To produce flutter the rate of stimulation must usually be raised to a point beyond the rate of the subsequent flutter: it must be raised to a point at which local block is to be anticipated. The impurity of flutter in its early stages and its instability in these stages (its instability in this stage is shown by its usual short duration) is accounted for if we conceive that during these stages the local barrier persists for a while, though it is diminishing. Consider, further, the nature of this local block when it is first produced and its influence on after-effects. Take the simplest instance. Let us suppose that the ring is receiving rhythmic stimulation at a high rate, and that these are passing in both directions through the ring (Fig. 6, 1) towards points *A* and *B*. A local block appears at *A* as the rate rises. This local depression of conduction will not block each wave. That is not our experience: it will delay the passage of most waves and will block the occasional one. So long as the wave goes through, its advancing border will meet the advancing border of the corresponding wave, travelling around the other limb of the circle, and the movement will then end. But if one wave is blocked, the tissue at the block will wait until the opposing wave reaches it: then, if it has passed from its refractory state, the opposing wave will proceed and the circus movement will become established. *But this event will only happen if the stimulus which creates the absolute barrier is the last artificial stimulus to enter the auricle.* So it will happen that if one wave in ten is blocked locally on the path of the circus that is to be, rhythmic stimulation may be withdrawn ten times, and on one occasion only will circus movement follow.

Now this is what happens when the auricle is stimulated. It is stimulated over and over again with rhythmic shocks of constant rate, and each time that stimulation ceases the heart returns to its normal rhythm; it is again stimulated at the same point and at the same rate, but, when the stimulation ceases, flutter becomes established for a longer or shorter while. Thus, we are able to explain on the basis of actual observation, first, why the

* As opposed to a unidirectional block such as Mines and Garrey cite. I do not say that unidirectional block may not be an occasional cause, but think that it is unnecessary to postulate it in explaining a circus movement following stimulation of the auricle, unless the rate of stimulation is less than that of the succeeding flutter.

wave on entering the ring is propagated in one direction* only; and, secondly, why the flutter after-effect of stimulation is so uncertain: to obtain success many repetitions of the same stimulation are required.

Take another simple instance. Suppose that the local block appears exactly on the opposite side of the circle to that at which the impulse enters. In this case the borders of the wave will meet in the region of block, and after-effects are hardly possible. Take another instance, namely, the one in which the block lies some way along one limb of the circle. Here again, unless conduction is much depressed throughout and the refractory period is very short, a temporary barrier will not establish circus movement. It seems therefore an almost necessary condition that the region of local block should lie near to the point at which the impulse enters. Perhaps this is why stimulation near the tænia terminalis is more effective than elsewhere, for in the sulcus is to be expected a natural line of block; that is, of course, purely a suggestion: the matter is not actually tested. But if the suggestion proves well founded, it will also lead us to understand why the short circuit between the superior and inferior cava is not adopted as the path in some instances of flutter. Once a circuit is established around both veins the absence of short circuiting between these veins is not difficult to explain, for it may be presumed that a wave crossing† would find the muscle on the opposite side still refractory from the passing of the last movement. The difficulty occurs only in understanding how the circus around both veins becomes *established*.

Mines found that once his circus movement in a ring of muscle was established, that a new and effective stimulus applied to the ring would usually bring the circus movement to an end. The reason given for this is that a new excitation entering the ring would render a portion of the ring refractory and alter the time relations of the re-entering wave. Long continued flutter can be brought to an immediate end by applying a few rhythmic shocks to the auricle: this has been witnessed where the flutter had lasted for a half hour or more, and where the possibility of coincidence with a natural ending was too remote to be considered possible. In this observation we have a precise parallel to that of Mines. But the method is not always efficacious: sometimes rhythmic stimulation may be continued for some while without effect. A reason for such failure is not difficult to give. One or more shocks must enter the muscle during the non-refractory phase: this phase for *any small* area of the muscle may be presumed to be of sufficient length to admit new impulses‡. Thus if rhythmic stimulation is continued for a few cycles a stimulus is certain to enter the muscle in its excitable phase and to yield a response. If such stimuli were applied to

* In one direction for any considerable distance.

† In either direction.

‡ If the circuit lasted from 0.1034 to 0.1714 of a second (see footnote, page 324) the non-refractory period would be at least as long as 0.0434 to 0.1114 of a second.

muscle lying in the central path, then we might perhaps always expect interference; but sometimes the stimuli will be applied, not to the central path but to the outlying regions, which receive and transmit centrifugal waves.* In such circumstances, such new ingoing waves as are propagated will meet the regular outgoing waves, and as these travel slowly and the excitable phases between them are not of long duration, it is clear that the central path may be completely shielded in certain circumstances from interference. It would appear that the treatment of auricular flutter in the human subject (which might be suggested) by attempting to break up the circus movement with induction shocks, cannot be regarded as offering hope of invariable success.

Another means by which, in experiment, flutter is sometimes brought to an end, is the application of a pair of contacts directly to the muscle. This happens† when the contacts are placed on the path which the central wave is suspected to be travelling. The same result will sometimes follow the lifting of the contacts. It is to be presumed that this is due either to a local change of temperature (as in the application of cold contacts) or to the effects of slight pressure or its relief. It is evident that, when the auricle is beating at the high rates prevailing in flutter, conduction is in a very sensitive state and changes easily: it is probable that any slight interferences may alter conduction, and if such an alteration occurs at a suitable point, interference with the circus movement and its cessation becomes intelligible.‡

If the views expressed are warranted, the establishment of flutter has as its essential underlying basis, a state of depressed conduction; this

* To state in any given experiment that the central path has been stimulated cannot often be justified. As a matter of fact, Mines did not always find stimulation of the ring effective in stopping the circus movement.

† Though it does not usually happen.

‡ In the present connection the possible influence of the pressure of contacts or temperature change induced by contacts upon curves which are instanced in the present article as examples of impure flutter, cannot avoid consideration. It seems perfectly clear, however, that this is not in any material degree the case. Thus if the direct lead is taken from outlying muscle, such as the superior cava, and disturbances are also witnessed in the main auricular cycles, these last can have nothing to do with the contacts placed directly on the muscle; moreover, a very local disturbance such as the contacts might be supposed to induce, would not be reflected in the form of the auricular complexes, the area of muscle involved would not be sufficient. When impure flutter prevails, a considerable area of muscle is almost always involved and involved in a very similar fashion. If the position of the contacts is changed, phenomena of a precisely similar order are witnessed at the new point, and this is so, irrespective of the degree of pressure with which the contacts are applied, or the length of time over which contact has been established. There is no hesitation in declaring the curves to be interfered with little, if at all, from this source. In taking curves of impure flutter, the contacts are laid on the muscle as lightly as possible, often they do not actually touch the auricle, the contacts being made by pericardial fluid which is held between contact and muscle by capillary attraction. It might also seem possible that some of the local variations which are witnessed are due to cooling of that portion of the surface of the heart which is exposed in the experiment. It is possible that local disturbances of conduction are more apt to happen in the exposed muscle than in that more deeply seated, and it is from this muscle that the records are most often taken. But they happen also in deeply seated muscle, muscle which is not so exposed, as observation shows. Thus, in leading from the left wall of the superior cava, or from the intra-auricular band, the contacts are pushed gently in between the aorta and auricle and make contact with muscle which is not exposed to atmospheric cooling. Irregular curves similar to those described are obtained from these regions of the auricle also. The underlying cause of local disturbances is to be found usually in the rate of the central movement, as the table on page 294 so clearly illustrates.

depressed conduction may be brought about in the first place by rapid beating in response to stimulation: subsequently it is maintained by the rapid action consequent on flutter itself. Thus, flutter becomes self-supporting and continuous. That flutter should be interrupted and brought to an end in the human subject, in which it is known to last in some instances for as long as six years, seems to require an alteration of the conduction state in the muscle, and treatment devised to that end appears to offer the best hope of success.

As I showed some years ago,² flutter may be brought to an end in the human subject by means of digitalis: it is converted into fibrillation, which subsequently often gives place to the normal rhythm as the effect of the drug wears off. It is tempting to conclude that this drug acts by depressing conduction in the auricular muscle. Actually, there is at present no direct evidence that digitalis has this action upon the ordinary auricular muscle of the mammal, though there is much indirect evidence to support the view. If this is indeed the method of its action, then much simpler and more efficient remedies at once suggest themselves. These are at present being investigated.

The theory of circus movement in the auricle as the underlying factor in flutter is based on the observations recorded in the second article of this series. It has now been re-examined in the light of further observations.

It has been seen (in the third and present article of the series) that the manner in which flutter begins, when it follows rhythmic stimulation of the auricle, is in accord with the theory: it is also in accord with observations upon simple rings of muscle. The reason why flutter may also follow faradic stimulation, as it frequently will, has not been alluded to, however. That is so, because reactions to this form of stimulation will be dealt with more fully when we come to study fibrillation. Suffice it for the moment to say that we are in possession of facts* which show that the full process set up by faradic stimulation is local, that the extreme frequency of response of the muscle beneath the contacts is not conveyed to the surrounding tissue for any great distance, but that the excitation waves are blocked in the muscle. Thus, the rate at which impulses reach the central path, supposing the appendix to be stimulated, is not a question of the rate at which the stimuli enter the latter. In other words, so far as the central path is concerned, faradic stimulation of the appendix has no very different influence to rhythmic stimulation at much lower rates.

In the third article of the series the way in which flutter ends has been described, and in that article, and in the present one, it is shown that the manner of these endings can be brought into line with the theory without difficulty.

* These observations, undertaken in conjunction with Dr. T. F. Cotton, will be described in their proper place.

In the present article it is shown that if the view is accepted that circus movement depends on lowered conduction, an extension of the same theory which involves no greater assumption than progressive and varying changes of conduction permits us to explain a very large number of curious disturbances which I have classed together as instances of impure flutter.

When impure flutter prevails, the rate of the central movement, as displayed by the auricular complexes in curves from lead *II*, is higher than when the flutter is pure.* The greater rate is often directly responsible for the impurity of the flutter: it throws a greater strain on conduction and brings about local block. The effects of a change in the rate of the central movement upon the events happening under direct contacts placed on the surface of the auricle have been more than once exemplified. There is perhaps no better example than that which the table on page 294 provides. In this table, which summarises a number of after-effects produced in a single auricle, the relation of fundamental rate to disturbance of the records is as definite as it well could be. Pure flutter was seen in this auricle at rates no higher than 460 per minute: at higher rates the flutter was impure on all occasions; and at the rate of 450 the flutter of this animal was unstable, passing very readily into an impure form: pure flutter established itself as a stable condition at a rate of approximately 370 per minute. This is one example of many others we possess of the close relation between the variety of flutter on the one hand, and the rate of the central movement on the other. The same theory of circus movement, ultimately disturbed, leads us up directly to a hypothesis explaining the nature of auricular fibrillation, which accords with the hypothesis already put forward by Mines and by Garrey, and with many facts collected by them and by other workers.

To conclude, the theory has so much direct evidence to support it; it enlightens and explains so many detailed observations; it points so clearly to the relation between a variety of disorders known to be quite closely allied: it accords so fully with the observations and conclusions of other workers, that any hesitation, which we may have felt in accepting it without reserve, dissolves.

SUMMARY OF OBSERVATIONS.

In this article conditions are described which sometimes follow periods of rhythmic stimulation of the auricle with induction shocks. They are termed collectively "impure flutter." The basis of "impure flutter" is considered to be the same as that prevailing in pure flutter, namely, a circus movement following a central path in the auricle. But in impure flutter the excitation spreads with less uniformity over the auricle from cycle to cycle.

The simplest disturbance is held to consist of local aberration of the centrifugal waves after they leave the central path; this local deflection or aberration produces local irregularities of beating, and these are compensated because the central rhythm is dominant and undisturbed.

* Presumably, in many instances of impure flutter a relatively short circuit is established.

These simple disturbances form a transitional series with similar but more complex disturbances, and lead up to the complex irregularities in which, so it is considered, the central circus movement is itself disturbed. The breaking up of the central circus movement is evidenced by lack of uniformity and sequence of the auricular complexes in lead *II*, and, as this proceeds, the curves come more and more to resemble those seen in clinical fibrillation of the auricle. It is considered that the latter becomes established when the central wave becomes submerged. In auricular flutter the excitation wave passes in equal times over equal stretches of auricular tissue: in impure flutter it does not appear to do so, and this change is attributed to the establishment of local obstructions or actual barriers (block) which deflect the wave along new and sinuous paths. When this process involves not only the outlying regions of muscle but also the path of the central wave, it is imagined that auricular fibrillation, as it is spoken of clinically, sets in.

The present observations confirm the conclusion that flutter results from the establishment of a solitary circus movement in the auricle, because the disorders described are explained if the depressed conduction, which is known to exist in flutter, is imagined to progress. Such progress is expected in that the rates of beating in impure flutter exceed those in pure flutter, and because such progress is witnessed when conduction is measured at different rates of response to artificial stimulation. The actual relation between the speed of movement in the mass of the auricular tissue and local disturbances is a curious one. A speeding up of the central wave which may result theoretically either from an increase in the conduction rate, or of its following a shorter track, throws a greater strain on the tissues supplied by the centrifugal wave, and produces in them greater disturbances in the spread of the wave as a whole from cycle to cycle.

The present observations serve to link up pure flutter, impure flutter and auricular fibrillation as a whole: they help to explain the close interrelation of all these conditions.

The theory of circus movement is discussed in more detail, especially in relation to the manner in which flutter establishes itself and is brought to an end.

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- ¹ GARREY. Amer. Journ. of Physiol., 1914, XXXIII, 397-414.
- ² LEWIS. *Heart*, 1911-12, III, p. 285; *ibid.*, 1912-13, IV, 171-220.
- ³ MINES. Journ. of Physiol., 1913, XLVI, 349-383 and Trans. Roy. Soc. of Canada, 1914, S. III, VII, 43-52.

A CORRECTION.

A statement has been made in a footnote to page 323 of this article to the effect that the length of the refractory state at any point of the auricular muscle is normally 0.06 of a second. In a longer footnote to page 324 the same time interval has been assumed. It is also assumed in a short footnote to page 327. The figure 0.06 of a second was taken from a paper published in this Journal (1920, vol. vii, page 152). Inadvertently the figure which was taken is that which represents the "fractionate contraction," *i. e.*, the phase during which the muscle is actually shortening at a given point: but the refractory period corresponds much more closely to this period of contraction plus that of relaxation. The period assumed for the refractory period should have been at least twice as long as the figure actually assumed. The conclusions of these footnotes are not tenable when the new figure is used.

Thus, it cannot be held that the duration of complete refractoriness of the auricle, beating normally, lasts probably only 1 or 2 hundredths of a second: its duration is certainly much greater. It is unsafe to state, as has been stated in the footnote on page 324, that it is not necessary to assume a reduced refractory period in explaining a maintained circus movement around the cavity: that may be, and very probably is, a necessary assumption.

With the exception of the conclusions of these footnotes, and what may prove to be an over-emphasis of depressed conduction, as the underlying basis of flutter, in the concluding remarks of the text, the error does not affect the conclusions of the article. The error was noticed too late to permit alterations of the footnotes in proof stage.

Explanation of plate figures. Each of the following figures consists of an electrogram from a direct lead and an electrocardiogram taken from lead *II*. Inter-intrinsic intervals and intervals separating auricular complexes, when inserted, are written in decimal points of a second horizontally. Intervals between auricular summits and corresponding intrinsic deflections in the direct leads are written vertically. The time lines represent fifths of a second. The standard used for the curves is 1 centimetre = 1 millivolt, in the case of lead *II*, and approximately 3.5 millimetres = 3 millivolts in the case of the direct leads. Some of the curves have been reduced a little. The horizontal lines are millimetre lines in the originals.

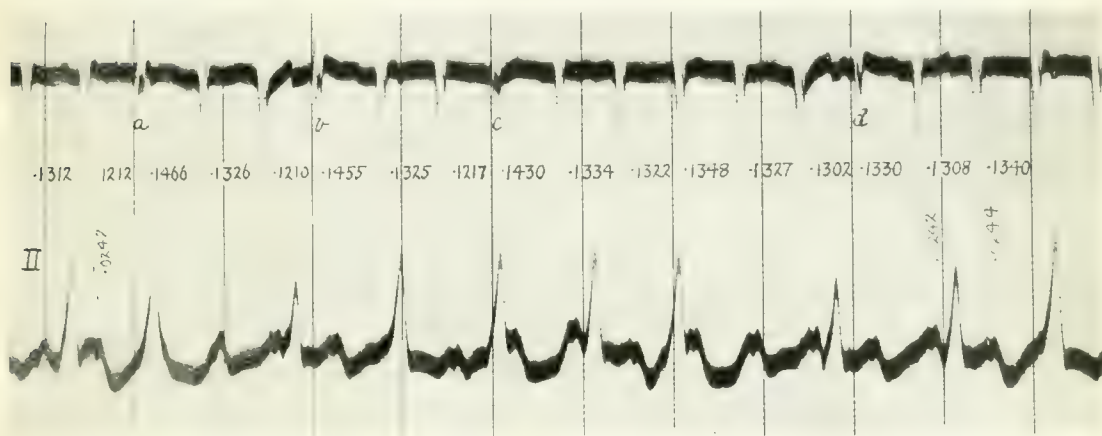


FIG. 7. (*Dog KQ. Record 14.*) From an after-effect lasting 26½ minutes: taken at 23 minutes. The direct lead was from the base of right appendix (*Z* contact below); the curve shows four disturbances, *a*, *b*, *c* and *d*.

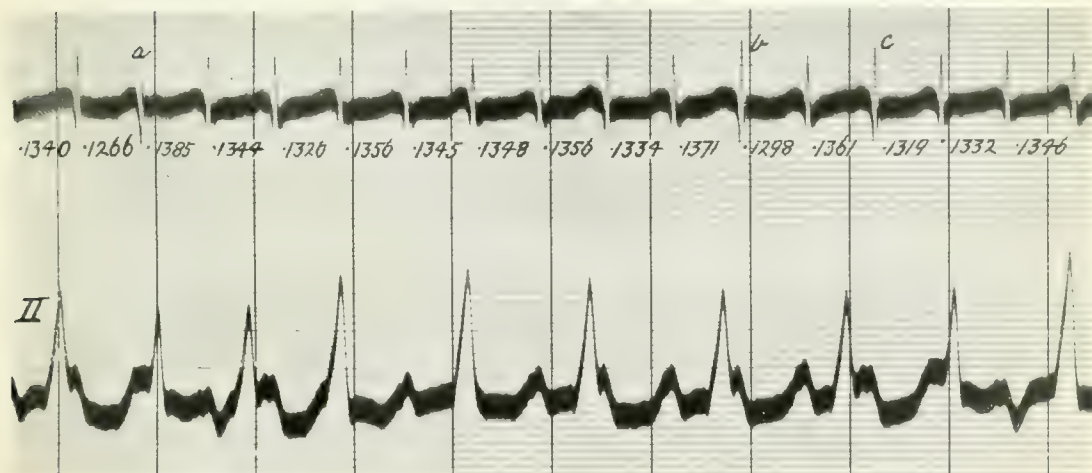


FIG. 8. (*Dog KQ. Record 15.*) From the same after-effect: taken at 25 minutes. The direct lead was the *S. V. C.* (*Z* contact below); the curve shows three disturbances, *a*, *b* and *c*.

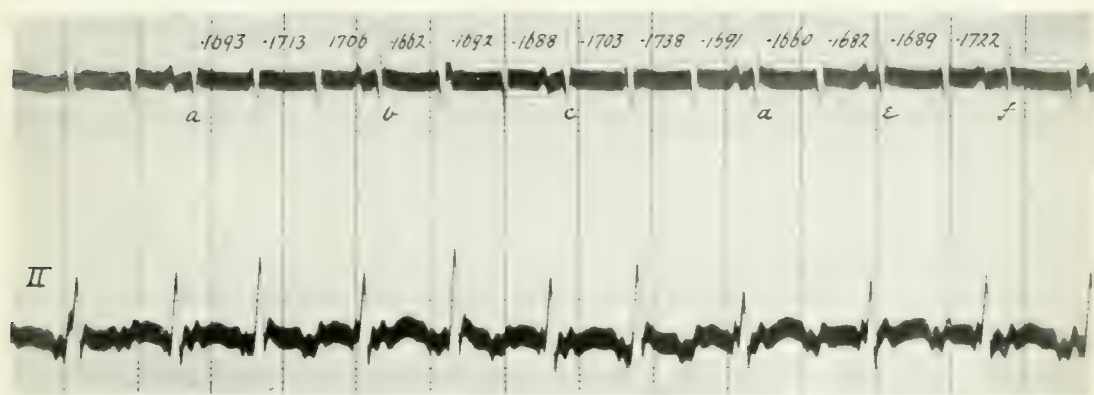


FIG. 9. *Lead KL. Record 10.* From an after-effect lasting nearly 5 minutes; taken at 47 minutes. The direct lead was from the base of the right appendix. Z contact used in I, V, C. The curve shows six disturbances *a* to *f*.

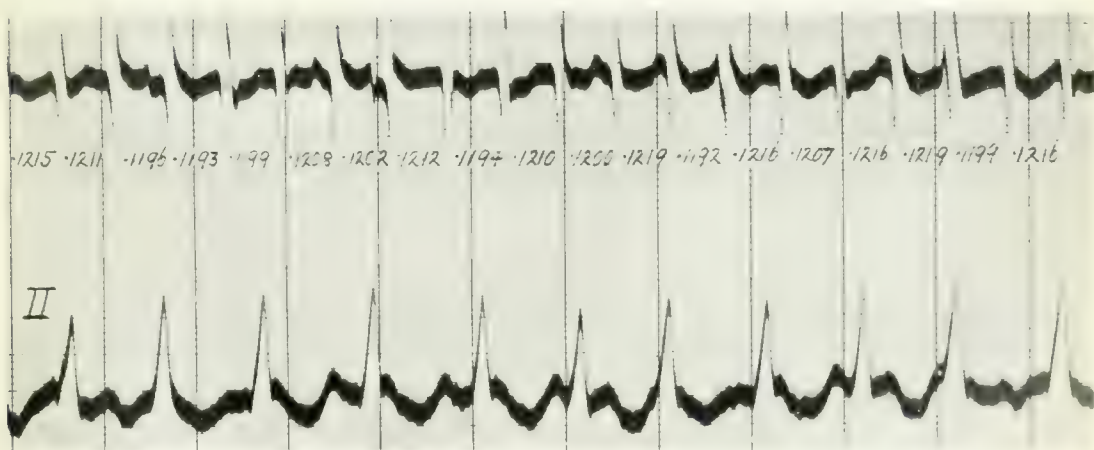


FIG. 10. *Lead KQ. Record 7.* From an after-effect lasting 20½ minutes; taken at 8½ minutes. The direct lead was the S. V. C. Z contact below 1; this curve shows no disturbance. The auricular complexes in the curve from lead II are distorted.

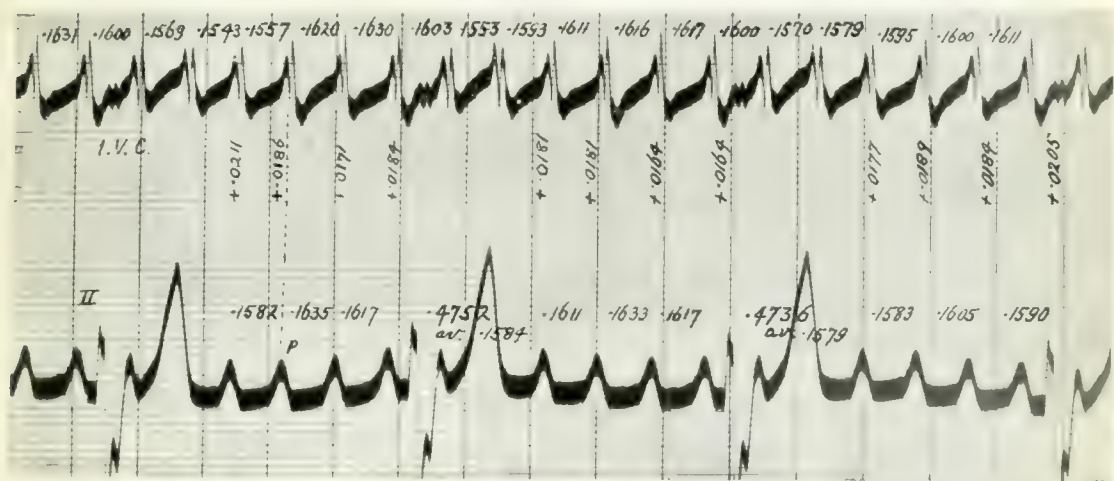


Fig. 11. (Dog J.P., Record 11.) From an after effect lasting 6 minutes, and fully described in a previous article (Part II). Taken after 4½ minutes. Direct lead from I.V.C. Pure flutter prevails. Compare with Fig. 12.

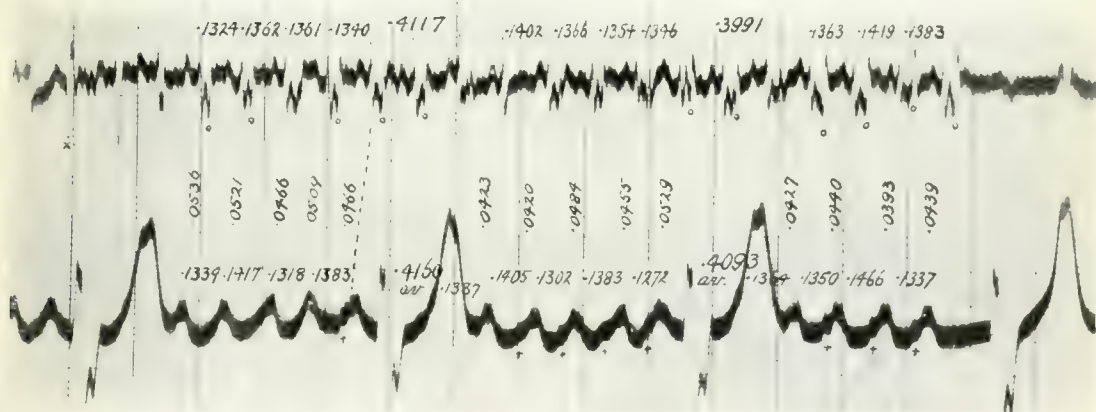


Fig. 12. (Dog J.P., Record 7.) From an after effect lasting 6 seconds. The direct lead is from the I.V.C.; the record shows impure flutter and distortion of the auricular complexes in lead II.

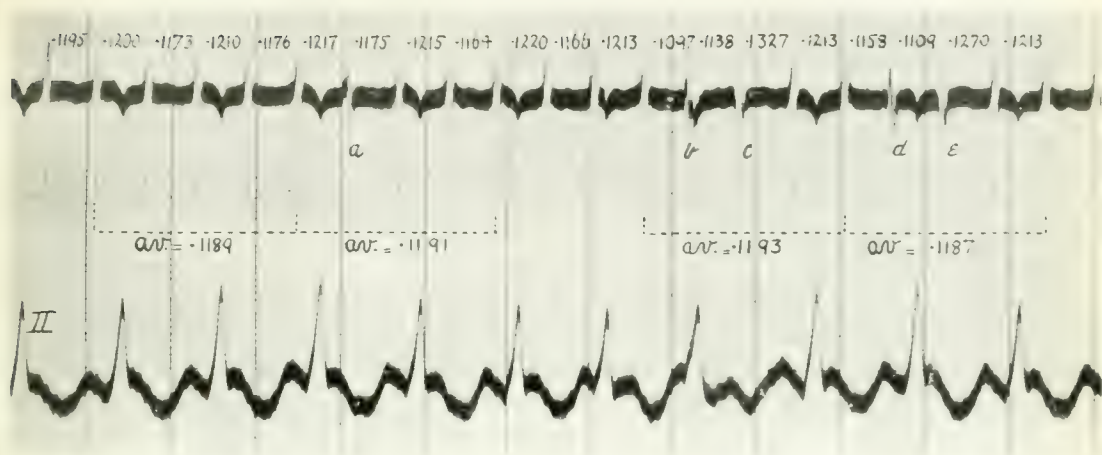


Fig. 13. *Dog KQ, Record 4.* From an after-effect lasting 26½ minutes, taken at 3 minutes. The direct lead is from the mid-caval region (Z contact below), and shows alternation and disturbances, *a, b, c, d* and *e*, all of which are compensated. The curve from lead *II* shows auricular complexes which are distorted.

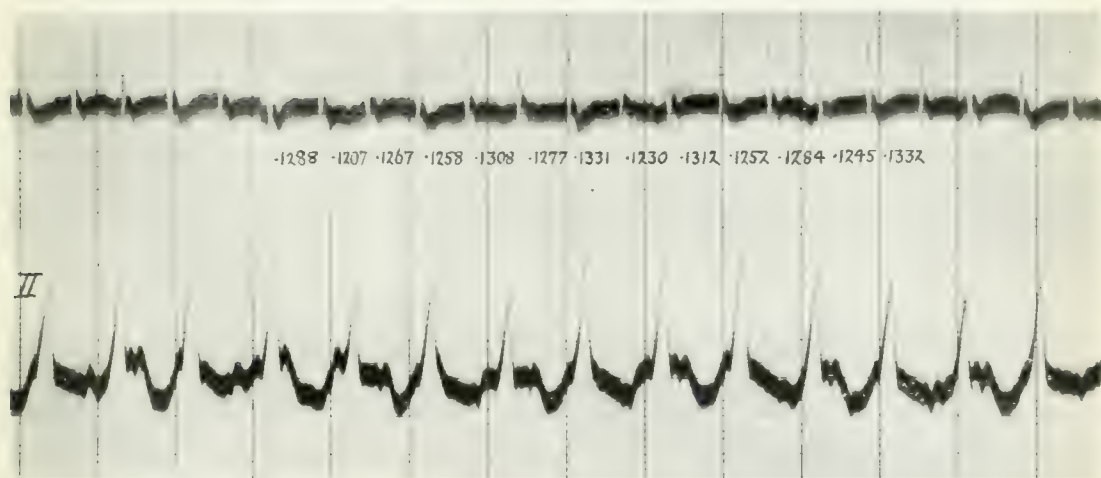


Fig. 14. *Dog KQ, Record 2.* From the end of an after-effect lasting 4½ minutes. The direct lead is from the I.V.C. (Z contact above). The record shows alternation in the curve from the direct lead and some distortion of the auricular complexes in the curve from lead *II*.

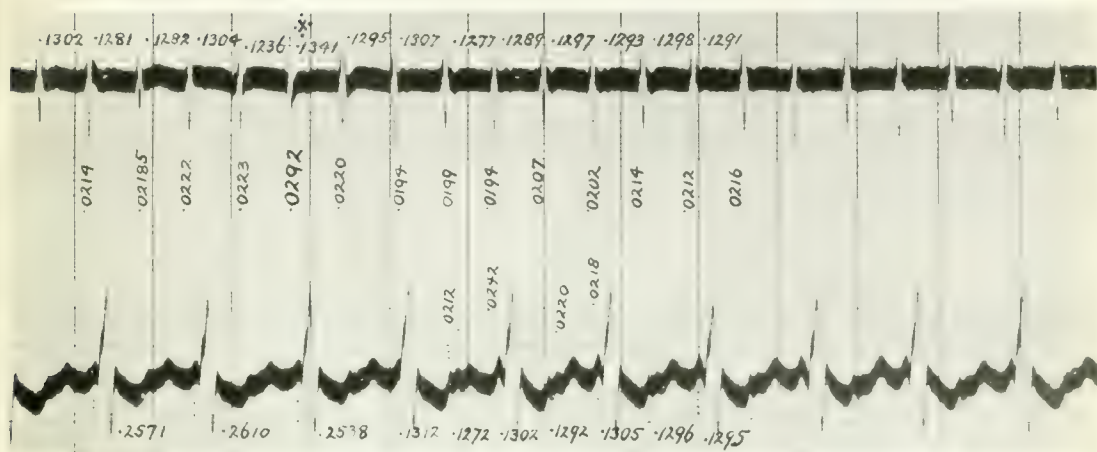


Fig. 15. Dog K 1. Record 21. From an after-effect lasting 20 seconds, taken at 13 seconds. The direct lead is from the mid-caval region (Z contact above); the curve shows alternation and a solitary disturbance (marked *). Above the electrocardiogram four figures are written vertically (.0212, etc.), these represent the intervals between the twin peaks of the auricular summit, as the dotted lines indicate. This animal was under the influence of eserine salicylate (1 milligram).

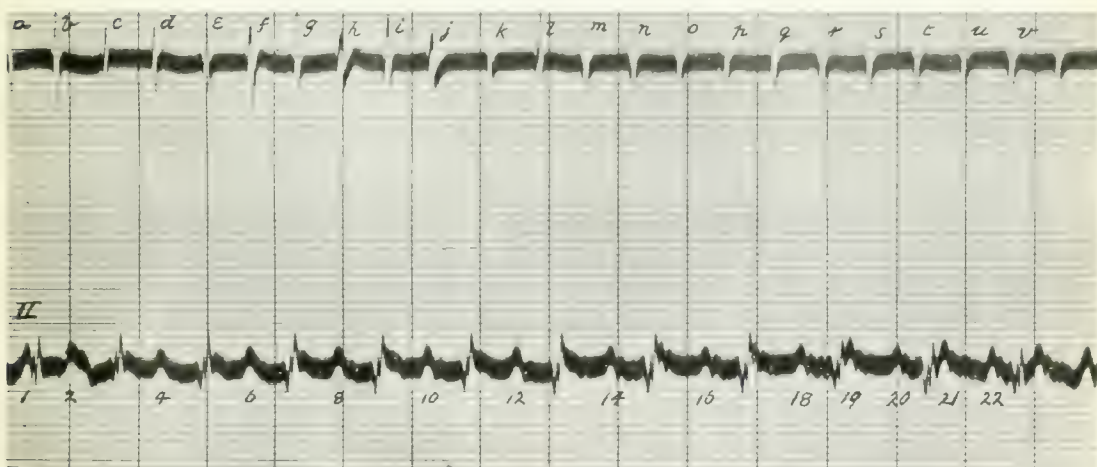


Fig. 16. Dog JS. Record 16. From an after-effect lasting 15 seconds, taken at 8 seconds. The direct lead is from the right appendix (Z contact below). The intrinsic deflections are lettered and the auricular complexes numbered to correspond with the chart, Fig. 1.

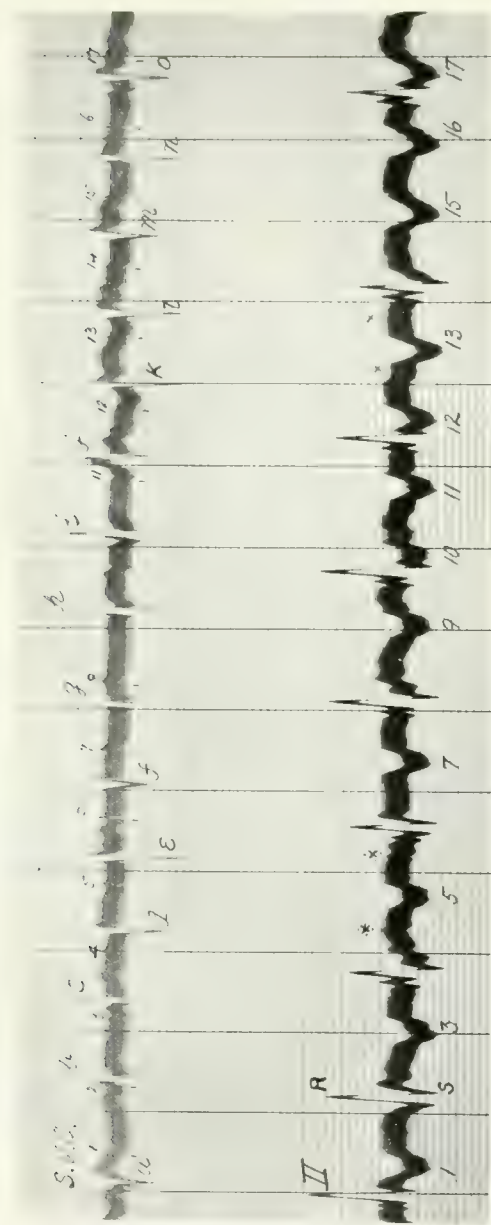
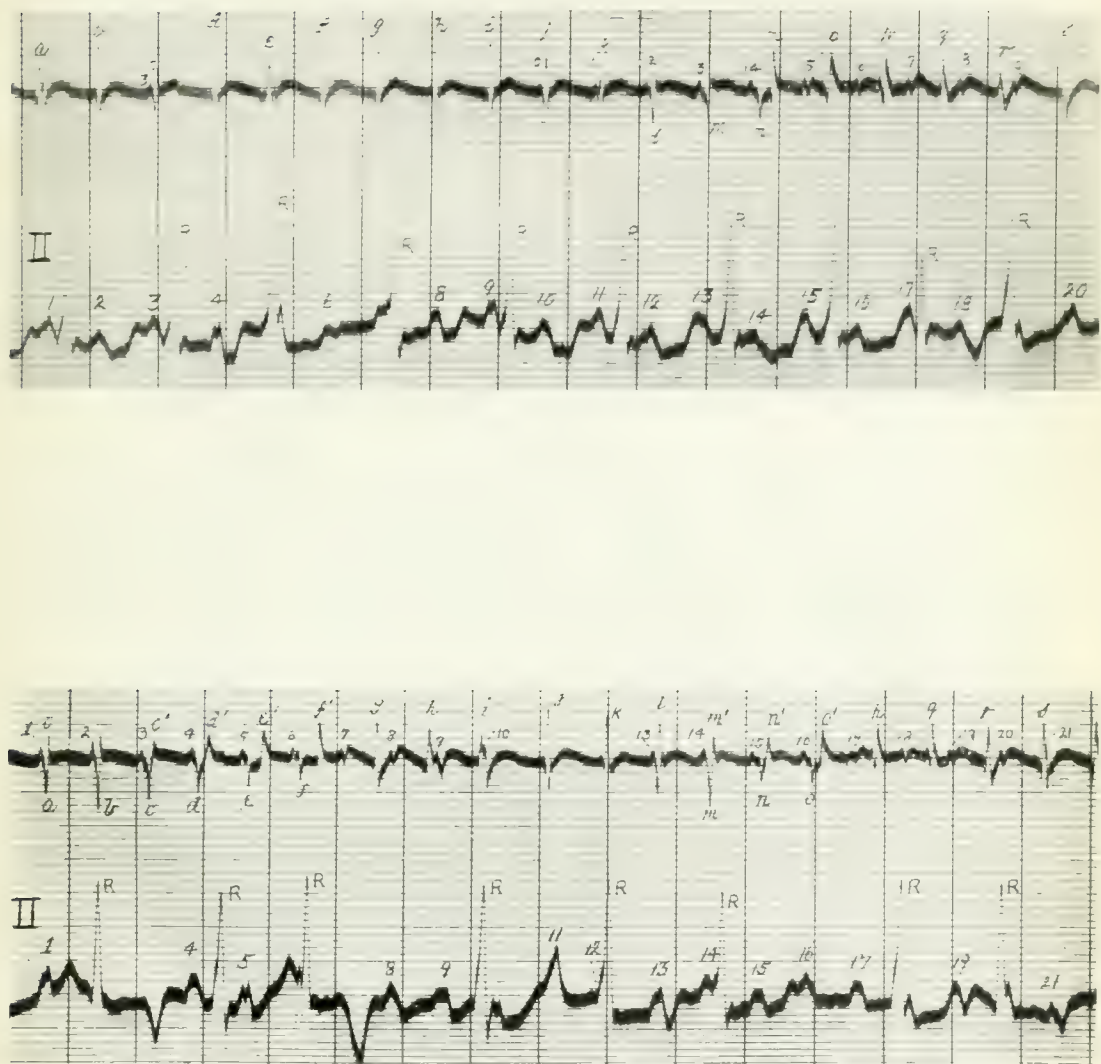


Fig. 17. (Dog K.L., Record 18.) From an after-effect lasting nearly 5 minutes, taken at 4 minutes. The direct lead is from the S, E, C, Z contact below). The intrinsic deflections are lettered, and the auricular complexes are numbered to correspond with Fig. 2. On the direct curve, small deflections corresponding with the auricular complexes, are numbered.



Figs. 18 and 19. (Dm J.J. Record 5 and 4.) From separate after effects lasting each a few seconds. The direct lead is from the body of auricle (C contact toward L.V.C.). The intrinsic deflections are lettered *a*, *b*, etc., and the auricular complexes are numbered to compare with Figs. 3 and 4. Small deflections on the curve from the direct lead are numbered to correspond with the auricular complexes of lead II.

THE RELATIVE EFFECTS OF RIGHT AND LEFT VAGUS NERVES ON THE HUMAN HEART.

By E. E. LASLETT.*

(Hull.)

DURING the past five years I have collected a long series of cases which showed a striking reaction to vagus pressure, with a view to examining into the significance of this test as a prognostic sign. In 108 of these patients it has been possible to determine the mechanism of the slowing by means of venous and radial curves. This paper gives an analysis of the results so far as they bear on the relative action of the right and left nerves.

Robinson and Draper⁵ found that the right nerve in man has a more marked effect on the rate of the heart, while the left has a greater effect on the conduction between auricles and ventricles. According to Ritchie,⁴ however, the inhibitory effect of the left vagus is less than that of the right on both the auricular rate and the junctional tissues. Cohn and Lewis¹ experimented on the dog. In order to eliminate the effect of auricular slowing, which Lewis believes may partly account for the difference between the two nerves in producing heart-block, they kept the auricular rate constant by rhythmic stimulation. They conclude that the right nerve chiefly controls impulse formation and the left nerve has a greater control of impulse conduction, but that the difference is quantitative rather than qualitative. Elsewhere Lewis³ has recently stated his belief that in their effect on impulse conduction the difference between the two nerves is more apparent than real.

While preparing this paper I have seen an abstract (*Medical Science*, June, 1920) of a recent paper by Kleeman.² Electrocardiographic records were made from 150 subjects. From an analysis of those who reacted to vagus pressure he concludes that the right nerve specially influences the sinus node, but it can also control the A-V node and bundle. Similarly, the left vagus can influence the sinus node.

The arbitrary standard of reaction adopted in the present series of observations is: (1) The reduction of auricular rate should be 2" or more, or in a few cases where the normal heart rate was more rapid a hundred per

* Undertaken on behalf of the Medical Research Council.

cent. slowing: (2) The production of a 2:1 or higher grade of heart-block. A few cases have been included which showed a long *a-c* interval, approaching two-fifths of a second or more without ventricular silences, as a result of vagal stimulation. Smaller increases of the *a-c* interval have been ignored (owing to the difficulty of accurate measurement of a venous curve) and because it was desired to utilise only those cases with a marked reaction.

Auricular Rhythm.

The following table shows the relative effects of the two nerves on the rate of the auricles. When the difference between the lengths of the standstill produced by each nerve is not more than one-fifth of a second the reactions have been recorded as equal.

TABLE I.

<i>R V</i> had a greater effect than <i>L V</i>	in 52 cases.
<i>R V</i> had a less effect than <i>L V</i>	in 39 ..
<i>R V</i> had the same effect as <i>L V</i>	in 16 ..
No slowing from either <i>R</i> or <i>L</i> (2:1 block from <i>L</i>) ..	in 1 case.
Total	108 cases

Many of the patients have been examined on several occasions at intervals, and it has been found that there is some considerable variation in the effect of vagus pressure on both impulse formation and conduction. The above table was compiled on the basis of the greatest effect obtained from each nerve. The right vagus produces the longer standstill more frequently than the left (expressed as a ratio it is 68:55), but the difference is not great. It has been not at all rare to obtain a pause 4.5 seconds long from the left vagus and the longest standstill observed—more than 8 seconds—was produced by this nerve.

According to Robinson and Draper,² with the right nerve the maximum pause initiates the onset of slowing, while with the left the maximum pause is delayed. I have not found this statement to be quite accurate. It depends on the length of pause. When marked slowing is induced by either right or left vagus the onset of the long standstill is usually abrupt with only slight preliminary lengthening of the preceding cycle.

Junctional tissues.

In twenty-nine of the patients a 2:1 or higher grade of heart-block was produced by vagus pressure, and in five others a marked increase of the *a-c* interval only. Ritchie found that any considerable effect on the junctional tissues was rare. The high proportion of heart-block reactions in this series may be accounted for partly, perhaps, by the greater age of many of the patients, but probably more by the fact that only cases showing a marked reaction to vagus pressure have been considered.

TABLE II.

Name.	Age.	Length of auricular cycle produced by vagus pressure expressed in $\frac{1}{2}$ sec.		Effect on junctional tissues.		Remarks.
		R Vagus.	L Vagus.	R Vagus.	L Vagus.	
PAT.	77	10	7.5	—	+ a-c	
BAR.	46	21.5	11.5	++ a-c	—	The long pause probably terminated by idio-ventricular beat.
BR.	65	7	8.5	3:1	2:1	Block produced much more readily by R than L
		9	16	+ a-c	+ a-c	
BU.	55	12	8	2:1	—	
CA.	68	8	5.5	—	2:1	
CR.	60	15	6	—	2:1	
DA.	62	8	10	—	2:1	
DIT.	76	5.5	Slight	4:1	—	
			Slowing	2:1	2:1	
DIX.	46	16	Slight	2:1	—	
FO.	86	7	Nil	2:1	—	
GA.	47	7	10.5	—	+ a-c	
GI.	57	5.5	Nil	2:1	—	
		7.5	—	+ a-c	—	
GO.	53	18	9	—	2:1	
GU.	70	7	11	—	3:1	
		10	8	—	2:1	
HU.	68	11	12.5	2:1	—	
JO.	43	7	Slight	2:1	—	
JOH.	68	Nil	12	—	2:1	
MA.	68	16	10.5	—	+ a-c	
MO.	57	7.5	15	2:1	—	
		7	7.5	2:1	—	
SE.	80	16	6.5	—	2:1	
SM.	81	5.5	5	—	2:1	
TH.	77	10	12	—	2:1	
TI.	38	Nil	7.9	—	2:1	
WA.	49	17	Nil	2:1	—	
WAR.	67	20	7	—	2:1	
WH.	50	7	Slight	2:1	—	R predominant in producing block.
		6	10	3:1	—	
		6	4.3	—	2:1	
WI.	50	18	7.5	—	2:1	
LE.	52	16	15.5	—	2:1	
HUT.	62	14	12	—	++ a-c	The beat terminating the long pause (L) probably idio-ventricular.
HUD.	64	12	6	2:1	—	Right nerve more powerful than left.
		9	6	2:1	—	
		7	7	2:1	2:1	
BR.	84	Nil	7.5	—	2:1	L predominant in producing block
		Slight	10	2:1	3:1	
		Slight	Slight	3:1	2:1	
HA.	47	Slight	8	—	3:1	
		6	10	—	+ a-c	
SML.	53	20	10	—	2:1	
CO.	42	6	Slight	2:1	—	
		7	7.5	2:1	—	

Right Vagus produced heart block in 14 patients and + — a-c interval in 1.

Left Vagus produced heart block in 19 patients and ++ a-c interval in 4.

Percentage ratio R : L = 15 : 23.

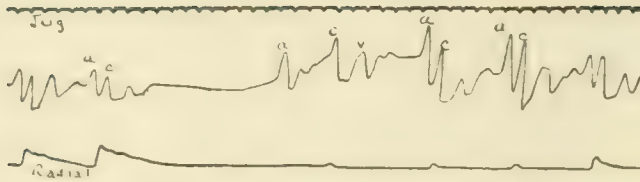


FIG. 1. *Bar.* Right vagus. The beat terminating the standstill may be idio ventricular, as *a* precedes *c* by an unusually long interval, namely, more than three-fifths of a second.

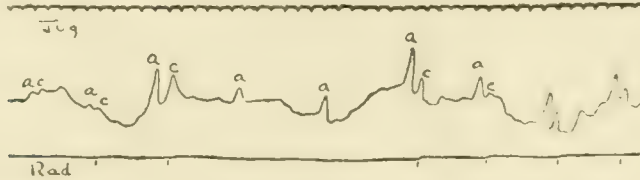


FIG. 2. *Wh.* Right vagus. To show 3:1 block. The *a-c* interval before the long pause is increased.

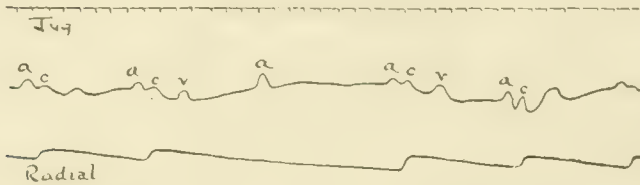


FIG. 3. *Mo.* Right vagus. To show 2:1 block.

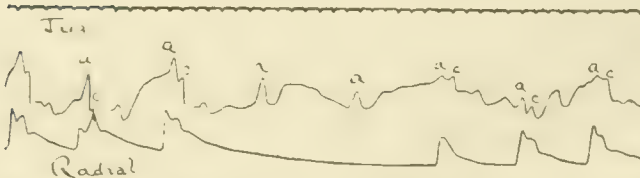


FIG. 4. *Gu.* Left vagus. To show 3:1 block. The auricular rate is only slightly slowed in this curve.



FIG. 5. *Ma.* Left vagus. A long standstill of the whole heart of 5 secs. duration. The *a-c* interval at the end of the pause is very slightly increased.

As already mentioned, Lewis believes that the difference between the effects of the two nerves on the junctional tissues may be partly explained by the fact that the right nerve slows the auricular rate more than the left, thus allowing a more complete restoration of conductivity in the bundle. Some of my curves show this clearly. Pressure on either right or left nerve would at one time produce slight slowing of the auricles with 2 : 1 block, and at another prolonged standstill with or without an increased *a-c* interval at the end of the pause. On the other hand, it will be found from the foregoing table that in many of the patients the effect on the auricular rate of the nerve which produces block may be as great as or greater than that of the other side. It must be remembered, however, that the method of stimulating the vagus in man is indirect and faulty as compared with the experimental method. The stimulus is not so likely to be evenly distributed to all the fibres as when the nerve is exposed. To what extent the reaction in man is affected in this way it is impossible to decide, but the totals for the two nerves seem to be sufficient evidence that though the left vagus has a greater effect on the junctional tissues than the right, the difference between them is comparatively small.

SUMMARY.

An analysis is made of 108 cases which showed a marked reaction to vagus pressure. It has been found that the difference between the effects of the two nerves is not very great.

The right nerve is predominant in controlling the auricular rate, but the left also retards the auricular rate to a very considerable degree. The ratio between the two may be expressed thus :— $R : L = 68 : 55$.

Similarly, the left nerve is predominant in the control of *A-V* conduction, but the right nerve has also striking control of impulse conduction. The ratio between the two is $R : L = 15 : 23$.

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AN ANALYSIS OF THE TIME-RELATIONS OF ELECTROCARDIOGRAMS.

BY H. C. BAZETT.

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Introductory.

IN a preliminary attempt (which requires considerable modification) to determine from blood-pressure records the relative influence of the heart action and of vaso-constriction, I suggested³ that it might be necessary to estimate the duration of ventricular systole for different heart rates. In order to obtain this information a number of measurements have been made of electrocardiographic curves, including some obtained by myself and a selection of curves from Dr. T. Lewis's collection, which he very kindly put at my disposal.

Electrical records have been preferred to mechanical, because it is easier to secure accuracy, and it has been shown by many workers that as a rule the electrical and mechanical changes correspond fairly closely. Lewis,¹⁷ in a comparison of the heart sounds with the electrical changes, found the first sound to commence 0.011 of a second to 0.039 of a second after the commencement of Q , while the second sound started either before or after the end of T but usually within 0.01 of a second of it. Wiggers,³¹ working with dogs, found the mechanical systole to commence 0.03 to 0.045 after the rise of R , and to terminate 0.034 to 0.048 after the end of T , so that as a rule the two changes corresponded in duration, but he found that adrenalin shortens the duration of the mechanical change more than the electrical, and under these conditions the ventricular contraction ended before the end of the T wave. In considering, therefore, the relative duration of systole and diastole, both electrical and mechanical records are useful, if these differences be allowed for.

Waller²⁹ gives the following values for the duration of mechanical systole with different heart rates, and it will be seen that almost exactly similar figures are obtained by calculation from the formula $\text{systole} = K \sqrt{\text{cycle}}$, where K has a value of 0.343.

Pulse rate.	Systole (Waller).	Systole (calculated).
50	0.37	0.376
60	0.34	0.343
70	0.32	0.318
80	0.30	0.298
90	0.28	0.28
100	0.27	0.266

It is one of the objects of this paper to establish such a relationship of systole to the square root of the heart cycle. Lombard and Cope²¹ have quite recently drawn attention to this from measurements of the carotid pulse, where the same condition seems to hold good, although by this method no account is taken of the period of rising tension. They give the duration of systole measured in this way as $\text{systole} = \frac{60}{k \sqrt{R}}$, in which R is the pulse rate and the value of k is 28.25 standing, 26 sitting, and 25 lying. After exercise they find systole relatively slightly longer giving a value for k of 27.5 standing. They also find the duration of systole is relatively longer in women. They suggest provisionally that the variations in systole under these different conditions may depend on differences in the venous return and filling of the heart in diastole. Their results are confirmed by the figures here reported, in spite of the difference in the method employed, and I had independently arrived at the same relationship of systole to the square root of the cycle before the publication of their results. A careful analysis of these time relations is demanded since some indication is afforded that they may supply a measure of the dilatation of a heart.

If Lombard and Cope's figures be taken, but systole be represented as a function of the heart cycle instead of the pulse rate, so that a factor is obtained for comparison with that suggested for Waller's figures, values for K of 0.275 standing, 0.298 sitting, 0.319 lying, and 0.282 for standing after exercise are obtained. These figures, neglecting the period of rising tension in the ventricles, are short by comparison with values obtained electrically. Edgren¹⁰ using an apex beat record found in man a duration of systole of 0.327 of a second with a pulse rate of 70 giving a value for K of 0.353 which is more directly comparable with electrical results, so that the differences between the two sets of figures appear to depend mainly on the inclusion or exclusion of the period of rising tension.

Weitz³⁰ has recently recorded the apex beat in man using a Franck capsule and optical methods. By this means he claims to be able to recognise auricular systole, the period of rising tension in the ventricle, the duration of the outflow, and the period of relaxation. The curves he gives resemble those shown by Wiggers and Dean³² for heart sounds combined with pressure changes.

From the curves given in his paper this method seems to supply a means of measuring accurately the time relations of the mechanical changes in the normal heart, and apparently he found it possible to do this also in the abnormal, though on *a priori* grounds one would expect this to be very difficult. He gives a considerable number of figures for normal hearts, and consequently they will be considered later in detail, together with the figures that I have myself obtained by the measurement of electrocardiograms from Dr. Lewis's cases or my own. Weitz gives in his tables the period of rising tension and the duration of outflow from the heart. The former he found to have a value of 0.035 of a second to 0.084 of a second, and this duration was the same whether the cycle was long or short. Its duration, however, was increased by high arterial tensions or by under filling of the heart, and decreased by low arterial tensions and by a big venous return of blood.

In the tables to be given later his two periods have been added together to give the total systole, so as to be comparable with the other figures, but his division into these two times is of considerable importance. The lower figures for rising tension time that he gives were found in children with low blood pressures, for adults the average figure was 0.06 of a second, and consequently the figures given by workers who have used the carotid pulse such as Lombard and Cope, can be compared with those obtained by other methods after the addition of this time. Thus Lombard and Cope's figures give a value of the systole, as measured in the carotid of 0.298 of a second for a pulse rate of 60, so that on adding 0.06 of a second to this the total systole of the heart would be 0.358 of a second (males). This figure compares remarkably well with an average duration of 0.368 for the same pulse rate when measured from a series of electrocardiograms, and the figure 0.363 is obtained for systole at such a pulse rate by a consideration of Weitz's figures. There seems to be then no doubt that figures obtained by various methods correspond remarkably well.

Method employed.

In the tables many of the figures obtained mechanically by Weitz are given for comparison with the electrical, which I have obtained from Lewis's curves and a few of my own. In obtaining my own records non-polarisable electrodes have always been used. The measurements were made by the use of a lantern or epidiascope, with which an enlarged image was thrown on to a screen made out of a large sheet of millimetre-ruled paper. In this way an enlargement was obtained in which about 30 to 50 centimetres corresponded to 1 second, and the duration of the curves could be read off on the paper with an accuracy of + or - 0.002 of a second for the more definite parts of the curves (*e.g.*, the *Q.R.S.* group), while they could usually be read with an accuracy of + or - 0.005 of a second in their other parts. In the selection of electrocardiograms only those curves were taken which were relatively easy to measure, and cases in which some fusion of two waves occurred were

avoided as far as possible. For this reason the time measurements were generally fairly definite. In all cases lead *II* was used, though in some cases measurements of lead *I* have been made for comparison, and a few of these figures are included in the tables, but are bracketed and have not been used in arriving at average figures. The duration of systole may be found longer or shorter in lead *I* as compared with lead *II*, but the average figures will be about the same in a series of cases, and the changes in the value of *K* under different circumstances seem to be much the same, whether the comparison be made using lead *I* or *II*. Consequently lead *II* has been used as the standard in preference to using the lead which gave the longest systole in any particular case, though the latter more laborious method may eventually prove to be the more useful. Very few measurements have been made of lead *III* records.

In most previous records of heart cycles, time measurements have usually been taken counting the cycle from the commencement of systole till the beginning of the next, but more consistent figures are obtained, from mechanical records as well as electrical, if the measurements are made from the beginning of diastole instead of systole. In arrhythmias both systole and diastole may be affected by outside influences, and they will both be more or less mutually dependent on one another, but the effect of diastole on systole will be the greater, since a long diastole will not only rest the heart but will also lead to its greater filling. A comparison of the two methods is on the whole in favour of the second and so bears out this contention, but in sinus arrhythmias there is little to choose between them, the change in pulse rate being in this case probably adapted to the venous return. In the tables the measurements for the cycle are always from diastole to diastole, except in the quotation of Weitz's figures.

It has only been possible to collect a few figures for normal women, but so many of these fall outside the range for normal men that they supply definite confirmation of Lombard and Copes' results. A few figures for the changes seen after exercise are also included; these exercise figures have been taken from curves obtained by Lewis and Cotton¹⁸ and used for their research into the shortening of the *P-R* interval with exercise.

In several cases the results of the examination of the same person on different dates are given to decide the amount of variation to be found in the same individual, and for similar reasons several consecutive cycles are given in detail in many of the records. On the other hand, in many cases a single row of figures represents the average measurements of several cycles. This has been almost invariably true when my own curves have been under examination, the object being to give the figures obtained from Lewis's curve a preponderating influence when arriving at general averages. Averages have always been used when the curves have been somewhat difficult to read accurately.

Blood pressures have been taken in my own cases using the auscultatory method and taking the change from the third to the fourth phase as the

indication of diastolic pressure. Blood pressure readings are also given by Weitz in many of his cases, and they are reproduced here but have been converted into millimetres of Hg instead of being expressed in centimetres of water, as in the original paper.

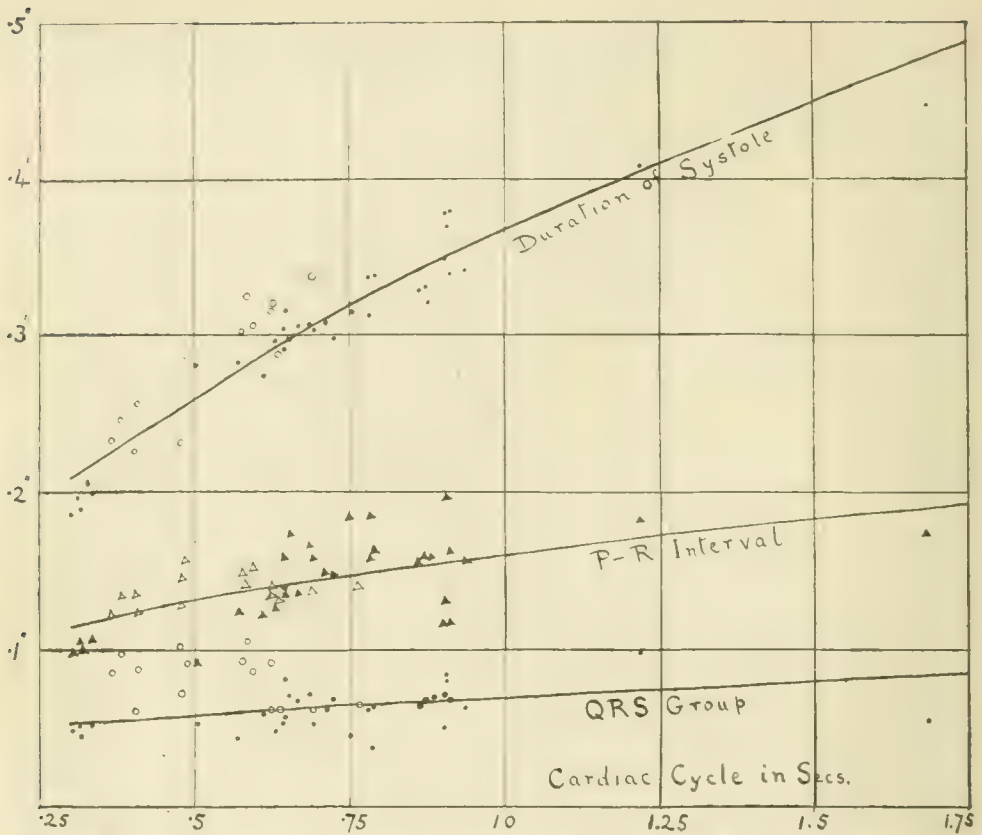
Results obtained.

The *normal figures* show a value of K *in men* of 0.368 for the electrical changes and of 0.363 for the mechanical. Variations of K between 0.342 and 0.392 are seen in the fifteen male subjects examined. The average values for the P - R interval, for the duration of the Q , R , S , group, and for the length of the P wave are respectively 0.149 of a second, 0.065 of a second, and 0.073 of a second.

The number of male subjects examined in detail is very small, but the normal value for K has been confirmed by a large collection of rougher measurements (made without enlargement) on various electrocardiograms in the literature. In these the values for K fall within the same limits, and also give an average figure between 0.36 and 0.37. In all about 35 other male subjects have been investigated as regards this point. The average duration for the P - R interval and for the Q , R , S , group correspond with those usually given, but that for the P wave is in agreement with the value of 0.08 of a second given by Kahn,¹⁴ and is rather short in comparison with the 0.1 of a second found by Einthoven¹¹ and the 0.095 of a second of Samojloff.²⁸

If the duration of systole is to be measured in relation to the cycle, it would seem reasonable to consider the other figures from the same standpoint. On plotting the figures out against the duration of the cycle, it is clear that there is a dependence of the one on the other, but these values remain little affected by changes in pulse rate compared with the duration of systole, and the changes observed are comparatively irregular. These irregularities are probably to be explained on the lines of Mines'²³ results on the effects of vagal and sympathetic stimulation on the conduction time in the frog's heart, *i.e.*, to fatigue (secondary to changes in pulse rate) competing with the direct effects of nerve stimulation. Thus it becomes more common to find a quickened rate of conduction with a slowing pulse in fatigued hearts, and this increased rate of conduction will often show itself in shortening the Q , R , S , group as well as the P - R interval. The duration of the P wave seems even more independent of the pulse rate, though this is probably not absolute, but this wave does not seem of sufficient importance to be worth more detailed consideration. It is only included here because it is lengthened in many cases of valvular disease. The Q , R , S , group is only slightly shortened with an increase in pulse rate.

The duration of the P - R interval varies to some extent with the pulse rate, but like the Q , R , S , group, it seems to be fairly constant and to be affected in the inverse direction by other factors such as fatigue. It will be seen, however, by reference to the figure, that in spite of the irregularities



Normal values for ventricular systole (electrical), the *P-R* interval, and the duration of the *Q.R.S.* group in men, including also five observations on infants, which form the five resting values at the extreme left of the curve.

Electrical Systole	Resting	●	} On the top curve.
"	After exercise	○	
<i>P-R</i> Interval	Resting	▲	} On the middle curve.
"	After exercise	△	
<i>Q.R.S.</i> Group	Resting	●	} On the lowest curve.
"	After exercise	○	

there is a rough dependence of the duration of both the *P-R* interval and the conduction time in the ventricle on the pulse rate. This may be expressed approximately by some formulæ such as—

cc note slip -
$$P-R = 0.06 - 0.10 \sqrt{\text{cycle}} \text{ and } Q.R.S. = 0.03 - 0.04 \sqrt{\text{cycle}}.$$

In suggesting provisional formula for the durations of the *P-R* interval and *Q.R.S.* group, the assumption has been made that in each there are probably two factors concerned, excitation and conduction. Hence the type of formula here given has been preferred, as easy to calculate and fitting the few available data as well as can be expected.

These formulæ make no pretence of any accuracy at present but merely provide a rough criterion for comparison. In the figure are included some values obtained in infants, in whom the distance the wave has to travel will be less, but there are only five of these charted, and these lie on the extreme left of the curve and rather below it, so that they do not really confuse the issue, while they are interesting for comparison, falling as they do, so close to the adult curve. For all ordinary pulse ranges the duration of these parts of the curve may be taken as practically independent of the rate, as is done generally at present, but the value of the *Q.R.S.* group is important, since, if systole is lengthened, it is clearly necessary to decide whether this has been brought about by a diminished rate of conduction. The value for the *Q.R.S.* group given here is perhaps on the short side of the real normal, since Lewis¹⁹ gives an average value of 0.0784 of a second for a pulse rate of 78 in seven unselected normal cases.

Normal women give figures very similar to those of the men, except that the duration of the ventricular complex is longer (0.342 as against 0.326 of a second), in spite of the fact that the average duration of the cycle is rather shorter (0.747 compared with 0.792 of a second). The average value for *K* for women is 0.399. For a duration of 0.747 of a second for the cycle the average systole for the male should be 0.318 of a second, so that this appears to be lengthened in women by 0.024 of a second. There is no evidence that this is due to any change in the conduction rate, which remains very constant, though there is some lengthening of the duration of the *P* wave by comparison with the men; no deductions should be made from this, however, since the *P* wave is often very difficult to measure accurately. In the table several figures are given also of measurements of lead *I* and one of lead *III*: some measurements were also made of these other leads in men, though they are not included in that table. The results were similar in the two cases. Though for most purposes measurements of lead *II* seem sufficient, in a few cases measurements of another lead may give the truer result. Thus in No. 71 of the tables lead *I* is probably the more accurate for both the duration of the *Q.R.S.* group and the duration of the ventricular complex.

The variations observed were greater in the women than in the men, but the general agreement with the long mechanical systole observed by Lombard and Cope is clear, and in certain cases this lengthening is very considerable. In No. 62 systole averages 0.3525 of a second for a cycle of 0.651 of a second, while the normal value for a man would be 0.297 of a second with an extreme upper limit of 0.32 of a second. For the women note has been taken as to whether the individual has taken much or little exercise, and where no remark is made the amount taken is medium, but no definite difference as a result of this factor can be determined from the figures available.

Infants show figures corresponding fairly closely with those of men, if the comparison is made with the short cycles found in men after exercise and if allowance be made for the slow conduction rate in men under these conditions. If the formulæ suggested for the determination of the normal length of the *P-R* interval and of the *Q.R.S.* group with varying pulse rate are correct, then these values for infants fall rather below the normal figures for men. Thus the *P-R* interval is 0.10 of a second as compared with a calculated 0.12 of a second, *Q.R.S.* is 0.049 of a second against a calculated 0.054 of a second, while the duration of systole is 0.212 of a second compared with a calculated 0.219 of a second for the same cycle in a man. Thus there seems to be a remarkable agreement between them and men, if allowance is made for a conduction time rather below the men's value. This allowance should certainly be made on general principles in consideration of the much smaller size of the heart in infants, since the importance of the length the wave has to travel is fully established¹⁹. The correspondence must then be considered as almost absolute, and it adds confirmatory evidence to establish the validity of the formulæ suggested. The figures given by Weitz for older children give similar results. These values for infants are also confirmed by rough measurements of the curves published by Krumbhaar and Jenks,¹⁶ which on analysis give an average value for *K* of 0.38.

Exercise in men produces a relative lengthening of systole as an after-effect, but these measurements of Lewis and Cotton's curves also show that the *Q.R.S.* group is little shortened with exercise, if at all, in spite of the change of pulse rate; it may even be lengthened. At the same time, by reference to the values obtained, and to their representation on the graph of the figure, it is clear that the *P-R* interval also, though shortened, has a longer duration than the normal for pulse rates of these frequencies. There is evidence, therefore, of some slowing of conduction as a result of fatigue, precisely similar to the effects obtained in the frog's heart by Mines.²³ The temporary lengthening of the *P-R* interval, which has been noticed to occur rather after exercise,¹⁸ would be of the same nature.

The lengthening of systole might be explained as being due to a slower conduction of the excitatory change through the ventricle in many of the figures given in the table, but this does not seem to apply to them all. Thus in No. 76 (c) the conduction rate is not very good, the *P-R* interval being lengthened, and if the difference in the conduction rate through the ventricle (an increase of 0.01 of a second in the *Q.R.S.* group) is deducted from the duration of systole, a value for *K* of 0.397 is obtained, that is to say, the whole increase in the systole might be explained by changes in the conduction rate. On the other hand, in No. 77 there is a similar lengthening of *K* after exercise, while the conduction time as judged both by the *P-R* interval and by the *Q.R.S.* group is shortened.

If the formulæ for the normal length of the *Q.R.S.* group be accepted, the normal value for the conduction time in any record can be calculated,

Bowen considered his results as due probably to an inability of the heart to empty an increased volume of blood against an increased resistance until adaptation had taken place. *I.e.*, he attributed it to a temporary heart dilatation. Aulo¹ obtained similar results.

Since these figures demonstrate that the value for K may vary considerably in any individual under conditions such as exercise, and yet that it has a fairly constant figure for different individuals under the same conditions, some values obtained in animals must be briefly considered, as they throw light on the factors which may affect it.

Patterson, Piper and Starling,²⁶ working with the isolated heart-lung preparation, found that not only the tension developed, but also the duration of the contraction was a function of the length of the fibres. With a rise in the systolic pressure there followed an increased heart volume and a "great prolongation" of systole without any change in the pulse rate. The increased blood pressure, by causing an improved coronary circulation, later led to a return to normal conditions. With an increased venous return the changes in heart volume and in the duration of systole were similar. Bowen's figures for the effect of exercise in man might then be explained on the basis of a temporary heart dilatation in concordance with these results on the isolated heart.

Patterson²⁷ also gives some figures for the effect of adrenalin and of carbon dioxide on the isolated heart, which show on analysis a great lessening of K after adrenalin, and a lengthening with carbon dioxide, and in agreement with this Patterson noted that the former reduced the mean volume of the heart and that the latter increased it. That the dilatation and change in K need not necessarily go together is shown by measurements of Figs. 26 and 27 in the paper by Starling and his co-workers, already referred to, where the fall in temperature is found to greatly increase the value of K without any corresponding dilatation being recorded. On the whole, however, their work suggests that K may prove a useful indication of the degree of dilatation of a heart.

The action of the vagus and sympathetic is also of importance. Hunt¹³ found that the vagus affected both systole and diastole, but the latter to a much greater extent, while sympathetic stimulation caused greater changes in the duration of systole in proportion to the change in pulse rate. Sympathetic stimulation, judged by his figures, lessens the value of K permanently, while section of the vagi increases K but only temporarily. An examination of his data, therefore, appears to show a lessening of the value of K for the mechanical changes with *both* sympathetic and vagal activity.

There is also no dearth of electrical records in the literature, from which values of K may be calculated. Buchanan² in 1909 and 1910 reported the effect of exercise using the capillary electrometer. Her data are in entire agreement with those already given for the effect of exercise, though

and if the figures given in the table for the effect of exercise be treated in this way and reduced values for K be obtained there is very little change in No. 77 as a result of the correction. In 77(b) of the tables K becomes 0.315, in (c) 0.349, and (f) becomes 0.406. But in Nos. 76 and 78 there is a much greater change. In 78 after correction the value for K remains between 0.362 and 0.367 throughout, but in 76 a corrected value of 0.369 before exercise is obtained, and K sinks as low as 0.329 in (d) and ends with a value of 0.355 in (f). A lengthening of systole then may, or may not, be associated with a change in the $Q.R.S.$ group. The time relations after exercise show big variations, as has been explained, but they may be approximately expressed by the formulae: Systole = $0.4 \sqrt{\text{cycle}}$, $P-R$ = $0.06 + 0.121 \sqrt{\text{cycle}}$, and $Q.R.S.$ = $0.03 + 0.08 \sqrt{\text{cycle}}$. Corresponding with the increased duration of the $Q.R.S.$ group there may also be some lengthening of the P wave. The number of cycles that have been measured is small, but a large proportion of the curves obtained are unsuitable for measurement owing to partial fusion of some of the waves.

Discussion of the results.

The figures given here might be open to criticism on the ground of their comparatively small number, when the importance of establishing standards is considered, but as they have been confirmed from various tables and figures in the literature, this criticism is not warranted, since some hundreds of figures have been employed.

Using mechanical figures of the apex beat Chapman⁶ determined the normal duration of systole for different pulse rates from 45 to 130, and his data agree very well with the principles here enunciated. The figures given by Donders⁹ in 1866 also agree quite well. Similarly, Bowen,⁴ using carotid records, has published a large number of figures for systole and diastole in man, and calculations from his data show a fairly constant value for K in different individuals, as well as reactions to exercise which are the mechanical counterpart of the changes shown in Lewis's and Cotton's galvanometer records. He found, it is true, that with light work the shortening of the cardiac cycle was almost entirely confined to diastole, but with heavy work, while this was also true at the commencement of the period, later shortening of the systolic period also occurred. Some of his figures may be given to demonstrate the similarity to those obtained from electrical records, and to show the gradual changes in K during exercise.

	<i>Cycle</i>		<i>Systole</i>		<i>K</i>		
Average of all	0.858	..	0.256	..	0.276	..	Resting.
Bowen's figures	0.600	..	0.266	..	0.343	..	Work started.
	0.488	..	0.212	..	0.304	..	Max. pulse rate.
for heavy work.	0.561	..	0.219	..	0.293	..	Work ended.
	0.769	..	0.244	..	0.278	..	Several minutes later.

see also Table p.

the value of K calculated from her figures is low, since to obtain definite points for measurement she used the commencement of R and the top of T . A few of her figures may be given for comparison with Bowen's mechanical records.

Buchanan, 1909. Subject, W. H. O. Effect of Exercise.

Cycle.	Systole.	K .	Remarks.
0.83	0.27	0.296	Resting. After a long run.
0.40	0.18	0.284	
0.34	0.16	0.274	
0.32	0.15	0.265	
0.55	0.22	0.296	
0.55	0.24	0.324	After a second long run.
0.34	0.18	0.309	
0.54	0.22	0.299	
0.62	0.26	0.330	
0.65	0.27	0.334	

Kraus and Nicholai¹⁵ have given a few figures for the duration of the ventricular systole measured electrically in normal people. Their figures show great variations even for the same pulse rate; and K , calculated from them, has values between 0.354 and 0.447; but their normals consisted of both soldiers and working women, and in consideration of the differences between the two sexes, these variations are readily explicable.

Examination of electrical changes obtained in animals brings out the same effect of the vagus on K , though the careful work of Mines²³ demonstrates the number of factors involved. How great may be the variations in K in an animal such as a dog, which may normally have an irregular heart action (due probably to variations in vagal tone) may be readily seen by reference to some of the curves published by Einthoven.¹¹ His Figure 3 shows three successive cycles of durations of 1.28 of a second, 0.76 of a second, and 0.63 of a second, with only a very slight change in the value of systole, so that K varies in this one record between 0.225 and 0.303. The average value for K in his figures of the dog's *E.C.G.* is about 0.265. He gives records of the effect of cutting both vagi; with intact vagi the pulse rate was 103, systole about 0.22 of a second and K about 0.29 of a second; after the section the pulse rate was 226, systole at least 0.18 (there is some fusion of the T and P waves), and K has a value of 0.35 or more. On the other hand, vagal stimulation diminishes the value of K (from 0.34 to 0.30 in his Figure 9).

Mines²³ published a number of careful observations of the electrical changes in the frog's heart, which throws some further light on the subject. Here again the square root function holds over a wide range of pulse rates, but only if allowance be made for the diminished rate of conduction in the ventricle resulting from fatigue at high pulse rates. In conjunction with Dorothy Dale⁷ he showed that nerve stimulation produced on the frog's heart a mixture of direct effects with others indirectly exerted as a result

of differences in the degree of fatigue of the heart, consequent on changes in the pulse rate. They also found that in the frog the durations of the electrical and mechanical changes did not necessarily undergo parallel changes, for the duration of the electrical change varied with the force of the mechanical contraction rather than with its duration. It is not clear how this observation should be correlated with the general agreement in the duration of these two processes in man, to which attention has already been drawn, but it is possible that discrepancies between the two series of figures in abnormal conditions are dependent on this factor.

Their figures show that when the direct effect of the vagus predominated the value of K was diminished, but that when the pulse rate was much altered there is an increase in K , once the total inhibition is over. With sympathetic stimulation the value of K (electrical) was somewhat increased. Their Figure 14 gives the following results: Before stimulation cycle 2.2 of a second with K 0.77, during stimulation cycle 1.44 of a second with K 0.81, and after stimulation cycles of 1.77 of a second with K 0.78, 2.08 of a second with K 0.88, and 2.14 of a second with K 0.85, and measurement of their Figure 15 produces similar results.

It seems clear, therefore, that the value of K may be much altered by vagal influences, at any rate temporarily, and that the vagus alters the mechanical and electrical changes in much the same way. But though increased vagal activity would decrease K by its direct action, this would be compensated for, at any rate to some extent, by the indirect effects resulting from the changes in pulse rate. Similarly, the changes in K during exercise may be explained: at first an increase in K as the result of diminished vagal action and a raised blood pressure, with a later return to a more normal figure when the sympathetic, after its longer latent period, comes into play, and improves the tone of the heart. On the other hand, it is by no means certain that the sympathetic affects the mechanical and electrical changes to the same extent. Mines found in the frog that, with the forcible contraction accompanying sympathetic stimulation, the electrical changes outlasted the mechanical. Wiggers³¹ found similar changes as the effect of adrenalin on the dog's heart, and the effect of sympathetic stimulation on the value of K shows similar differences (cf. Hunt and Mines). In the case of this also, as in the vagal action, there must arise indirect influences, secondary to the circulation changes, tending to neutralise the direct effect, and this will make for constancy. That the normal changes in pulse rate are produced in man by the combined action of the vagus and sympathetic seems clear from the work of Favill and White¹² on an individual who was able to produce a voluntary acceleration of his pulse. Rough measurements of the electrical records they give show a value for K of about 0.38 under normal conditions, of about the same after exercise, and of 0.413 when the heart was voluntarily accelerated. These figures, are, however, probably considerably affected by the fact that with the acceleration there was a marked rise of blood pressure.

The condition of the heart after exercise has been investigated by many different methods, and it seems clear as a result of X-ray examination—Dietelen,⁸ Nicholai and Zuntz,²⁵ Williamson³³—that soon after the exercise is over any increase in the mean volume of the heart that may have been present passes away, so that its size is normal or below normal. This, no doubt, corresponds with the development of a subnormal blood pressure, and a small pulse pressure. It is with the conditions of this period that the electrical changes after exercise have to be correlated. It has already been pointed out that the lengthening of systole may be due to a diminished rate of conduction, and that if allowance is made for this systole, is even shorter than normal, except in the case of No. 77. Two explanations seem possible: systole is increased after exercise solely as the result of fatigue and slowed conduction, this increase being to some extent neutralised by an opposite effect produced by the lowering of the blood pressure and a diminution of the size of the heart. On this theory the changes in No. 77 must be attributed to the masking of the true duration of the *Q.R.S.* group in lead *II*, or else to this subject being one of those rare individuals who show dilatation of the heart for some time after exercise (cf. Williamson). The other alternative is, that the whole method of judging systole after making allowances for changes in conduction time is wrong. The figures of many cycles have been examined, taking other periods, *e.g.*, the end of *S* to *T* and top of *R* to *T*, but none show such consistent results with changes in pulse rate as those of the whole ventricular complex, and it is conceivable, though unlikely, that the length of this complex is independent of the length of the *Q.R.S.* group.

The difference seen between men and women in the electrical changes, confirming those found by mechanical methods by Lombard and Cope, must be genuine and is difficult to explain. For women the value of *K* varies between 0.36 and 0.44 with an average of about 0.4, but the difference does not at present appear to be definitely dependent on the degree of training of the individual. Further data on the effect of training are at present being collected by another worker in this university.

This difference between men and women might be explained by the assumption that those with the larger value of *K* have hearts which are unable at the time of examination to do their work even at rest without a certain amount of dilatation, in accordance with the theories developed by Starling and his co-workers. If this is true, one would expect that such hearts would be unable to increase their output per beat during the performance of extra work to any great extent, and their circulation rate would then be determined mostly by changes in the pulse frequency. In the extreme case the results would fall into line with the figures often obtained in untrained individuals by methods for estimating the circulation rate. Bainbridge² has analysed some of Boothby's and Lindhard's figures from circulation rate experiments by respiratory methods, and both Boothby and Frau M. would come in this class of untrained persons who increase their output

per beat with very little exercise. If the theory is true, then such people should show a large value for K when at rest. The results would also agree with the fact that for equal body weights the size of the heart in women is on the average definitely smaller. This is very clear from Muller's²⁷ figures for the size of the heart in man, which show the weight to be about 10 per cent. greater in men than in women. In the larger body weights the difference is even greater. Thus for weights between 65 and 70 kilos, in men the average figure for the heart was 335.9 gm., and in women 262.9, and in this group the average age of the men and women was about the same. Such differences in the relative size of the body and heart would be almost bound to lead to differences in function.

The figures that have been quoted suggest that the duration as measured by the value of K mechanically and probably also electrically, may be an indication of the degree to which the heart dilates in order to maintain the circulation. If, therefore, this proved to be a measure of dilatation, its clinical importance would be very great. Consequently, this paper aims at establishing the normal limits of these relations, and a comparison between these figures and those found in pathological conditions will be published later. Recently Meakins²⁸ has brought forward figures which show that the duration of the electrical systole is much increased in patients with large hearts, especially those with preponderance of the left ventricle, but his figures have not taken into consideration changes in pulse rate, nor do they give sufficient data to decide the questions here raised.

CONCLUSIONS.

1. The duration of the ventricular complex in an electrocardiogram is in the normal heart a function of the pulse rate, and may be determined by the formula: $\text{Systole} = K \sqrt{\text{cycle}}$. The normal value for K is 0.37 for men and 0.40 for women. These observations confirm the relationships found by Lombard and Cope with mechanical records of the carotid pulse.

2. The value of K is somewhat increased after exercise, and this is often associated with a slow rate of conduction in the ventricle. The value of K may also be altered by an absence of the normal balance between vagal and sympathetic activity, an increase in vagal inhibition causing a lower value for K , and *vice versa*. The value for K is consequently particularly increased at the commencement of exercise, but any such effect appears to be temporary.

3. Evidence is also brought forward suggesting that K may prove a measure of heart dilatation, and that the changes seen in exercise may be partly due to this factor.

In conclusion, my thanks are due to Dr. T. Lewis for so kindly placing his material at my disposal, and to some of the women students in the university for their co-operation in the collecting of curves of normal women; also to Miss Buchanan for very kindly drawing my attention to some of the literature of the subject.

TABLES.*

Normal children. Mechanical (Weitz).

No.	Age and sex.	Cycle.	Systole.	P.	P.R.	Q.R.S.	K.	Remarks.
1	3 M.	0.385	0.215				0.346	
2	3 M.	0.435	0.245				0.371	B.P.† 90/63
3	3 F.	0.473	0.255				0.371	B.P. 78
4	5 F.	0.526	0.270				0.373	
5	11 F.	0.530	0.295				0.407	
6	6 F.	0.576	0.301				0.398	
7	6 F.	0.589	0.264				0.344	
Averages		0.502	0.263				0.373	

Normal children. Electrical.

No.	Age and sex.	Cycle.	Systole.	P.	P.R.	Q.R.S.	K.	Remarks.
8	1 day M.	0.303	0.184	0.036	0.099	0.047	0.334	Same child.
9	2/52 M.	0.318	0.189	0.041	0.104	0.045	0.336	
10	1 12 M.	0.336	0.201	0.052	0.106	0.051	0.347	
11	2 12 M.	0.318	0.206	0.072	0.100	0.051	0.365	
12		0.503	0.282	0.052	0.091	0.052	0.397	
Averages		0.356	0.212	0.051	0.100	0.049	0.362	

Normal men. Mechanical (Weitz).

No.	Age.	Cycle.	Systole.	P.	P.R.	Q.R.S.	K.	Remarks.
13	18	0.570	0.295				0.390	B.P. 120/77
14	20	0.575	0.310				0.408	
15	21	0.594	0.259				0.336	
16	18	0.730	0.305				0.357	
17	21	0.732	0.347				0.405	
18	33	0.736	0.316				0.368	
19	24	0.743	0.302				0.350	B.P. 124/88
20	18	0.745	0.305				0.353	
21	18	0.752	0.292				0.336	
22	18	0.755	0.320				0.368	
23	19	0.757	0.339				0.389	
24	38	0.778	0.342				0.388	
25	21	0.820	0.315				0.348	
26	18	0.821	0.326				0.360	
27	18	0.822	0.312				0.344	
28	23	0.825	0.335				0.368	B.P. 111/74
29	29	0.840	0.330				0.360	
30	18	0.865	0.340				0.365	
31	28	0.887	0.347				0.368	
32	19	0.905	0.345				0.363	
33	18	0.991	0.331				0.333	
34	19	1.443	0.378				0.352	
Averages		0.804	0.322				0.363	

* In the tables all the normal women are my own cases except No. 63, and in the other tables only those patients have been examined by myself, in whom a record of the blood pressure is combined with one of the electrical changes.

† B.P. = blood pressure, systolic or systolic and diastolic.

Normal men. Electrical.

No.	Age.	Cycle.	Systole.	P.	P. R.	Q. R. S.	K.	Remarks.
35	27	1.220	0.414	0.090	0.182	0.098	0.374	Same subject on different occasions.
36	27	0.792	0.338	0.070	0.131	0.064	0.378	
37	27	0.938	0.342	0.054	0.156	0.063	0.352	
38	27	0.912	0.339	0.064	0.162	0.069	0.356	
39	28	0.904	0.350	0.090	0.131	0.050	0.368	
40	25	0.647	0.291	0.055	0.137	0.056	0.361	Same subject.
41	18	0.714	0.308	0.065	0.138	0.062	0.365	
42	18	0.686	0.306	0.073	0.166	0.072	0.369	
43	18	0.666	0.306	0.083	0.136	0.068	0.375	
44	Boy	0.571	0.282	0.075	0.124	0.043	0.373	
45	36	0.632	0.296	0.071	0.126	0.048	0.372	Same subject.
46	36	0.907	0.370	0.092	0.196	0.082	0.388	
47	36	0.693	0.309	0.076	0.159	0.053	0.371	
48	28	0.647	0.304	0.080	0.159	0.054	0.378	
49	28	0.725	0.294	0.067	0.148	0.068	0.348	
50	28	0.783	0.312	0.074	0.159	0.061	0.352	Same subject on two occasions, and on 2nd in different positions.
51	28	0.880	0.321	0.059	0.159	0.069	0.342	
52	28	0.864	0.329	0.076	0.156	0.065	0.354	
53	28	0.873	0.331	0.049	0.159	0.067	0.352	
54	—	0.654	0.298	0.082	0.174	0.071	0.368	
55	30	0.612	0.274	0.085	0.122	0.059	0.350	B.P. 116/75
56	14	0.928	0.378	0.052	0.117	0.072	0.392	
57	14	0.932	0.379	0.055	0.117	0.080	0.392	
58	36	0.747	0.332	0.079	0.139	0.058	0.384	
59	40	0.705	0.310	0.082	0.163	0.071	0.369	
60	34	0.783	0.337	0.080	0.186	0.037	0.381	B.P. 128/76* B.P. 108/80
61	—	0.751	0.315	0.080	0.186	0.045	0.363	
62	—	1.660	0.443	0.071	0.173	0.054	0.344†	Same subject.
Averages		0.792	0.326	0.073	0.149	0.065	0.368	

Normal women. Mechanical (Weitz).

No.	Age.	Cycle.	Systole.	P.	P. R.	Q. R. S.	K.	Remarks.
55	22	0.555	0.265				0.356	B.P. 108/81
56	33	0.702	0.315				0.374	
Averages		0.628	0.290				0.365	

Electrical.

No.	Age.	Cycle.	Systole.	P.	P. R.	Q. R. S.	K.	Remarks.
57	21	0.832	0.403	0.105	0.164	0.077	0.444	B.P. 115/75
58	23	0.723	0.372	0.095	0.137	0.085	0.438	B.P. 116/70
59	26	0.638	0.305	0.080	0.136	0.069	0.382	Sedentary.
60	23	0.787	0.332	0.107	0.175	0.067	0.374	B.P. 104/60
61	30	0.817	0.327	0.109	0.185	0.060	0.388	Athletic in past.
62	21	0.885	0.394	0.088	0.133	0.078	0.419	B.P. 108/65
63	—	0.645	0.353	0.080	0.122	0.076	0.440	Athletic.
64	—	0.657	0.352	0.078	0.127	0.089	0.434	B.P. 94/66
65	—	0.569	0.304	0.073	0.154	0.067	0.403	Sedentary.
66	—	0.680	0.363	0.068	0.092	0.065	0.381	B.P. 108/78
67	—	0.935	0.356	0.077	0.121	0.048	0.364	Sedentary.
68	—	0.845	0.336	0.068	0.116	0.053	0.366	Lead I
								Sinus arrhythmia.

* This case gave a mechanical systole by a carotid record of 0.24 of a second, with a cycle of 0.732 and K 0.281 for comparison with Lombard and Cope's results.

† Athlete with slow pulse (Lewis's¹¹ "Clinical Electrocardiography," Fig. 34).

Normal women. Electrical—continued.

No	Age.	Cycle.	Systole.	P.	P.R.	Q.R.S.	K.	Remarks.
64	22	0.662	0.304	0.080	0.137	0.074	0.374	B.P. 105/75. Seden- tary.
		(0.639)	(0.308)	(0.072)	(0.134)	(0.062)	(0.386)	
65	23	0.725	0.345	0.102	0.161	0.081	0.405	B.P. 110/76. Past Athletics.
		(0.740)	(0.349)	(0.057)	(0.124)	(0.086)	(0.406)	
66	53	0.858	0.368	0.106	0.170	0.063	0.397	Heart probably normal.
67	20	0.619	0.314	0.085	0.136	0.042	0.399	
68	22	0.681	0.336	0.077	0.162	0.072	0.407	B.P. 124/78
69	22	0.645	0.327	0.070	0.120	0.068	0.406	B.P. 120/70. Knee an- kylosed since age 7. Mech. syst. by carotid K, 0.302
70	20	0.825	0.360	0.083	0.156	0.082	0.400	
71	20	0.677	0.323	0.096	0.176	0.046	0.393	Difficult to read.
		(0.618)	(0.330)	(0.065)	(0.139)	(0.073)	(0.424)	
72	23	0.596	0.312	0.087	0.164	0.079	0.404	Athletic. Hockey 1 hr. previously.
73	20	0.654	0.346	0.079	0.137	0.058	0.427	Athletic.
74	20	0.808	0.325	0.077	0.140	0.054	0.363	Athletic.
		(0.815)	(0.340)				(0.377)	Lead I.
75	24	0.842	0.366	0.066	0.146	0.054	0.398	Athletic.
		(0.130)	(0.386)	(0.109)	(0.159)	(0.057)	(0.363)	Lead I.
		(0.927)	(0.333)			(0.054)	(0.356)	Lead I I I.
Averages		0.747	0.342	0.085	0.142	0.067	0.399	

Normal men. Effect of exercise. Electrical.

No	Cycle.	Systole.	P.	P.R.	Q.R.S.	K.	Remarks.
76a	0.648	0.316	0.095	0.138	0.081	0.392	Before work.
b	0.368	0.234	0.086	0.123	0.085	0.386	½ minute to 1 minute after.
c	0.382	0.247	0.097	0.134	0.097	0.400	1 minute to 2 minutes after.
	0.409	0.257	0.093	0.125	0.087	0.402	
d	0.478	0.270	0.078	0.128	0.101	0.390	3 minutes after.
e	0.487	0.287	0.112	0.156	0.091	0.412	5 minutes after.
f	0.578	0.303	0.088	0.148	0.093	0.398	7 minutes after.
77a	0.765	0.306	0.074	0.140	0.065	0.350	Before work.
b	0.479	0.232	0.068	0.144	0.072	0.335	After.
c	0.405	0.227	0.089	0.134	0.060	0.357	After.
d	0.636	0.288	0.067	0.131	0.062	0.351	After.
e	0.627	0.320	0.074	0.140	0.062	0.404	After.
f	0.690	0.339	0.076	0.138	0.061	0.408	After.
78a	0.596	0.307	0.109	0.151	0.086	0.397	9 minutes after work.
b	0.585	0.325	0.117	0.141	0.105	0.425	11 minutes after.
c	0.623	0.316	0.102	0.135	0.092	0.400	13 minutes after.

Average value for *K* before exercise (two obs. only) 0.371 and lengthening of *K* 1 to 2 minutes after cessation of exercise. Values for No. 76 *b* and *c* rather uncertain owing to occasional fusion of the *P* and *T* waves.

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